INTERNATIONAL ASSOCIATION FOR DENTAL RESEARCH

Scientific Proceedings of the Ann Arbor Section

May 10, 1928

U. G. RICKERT, A.M., D.D.S., *Editor*

Joint meeting of the Ann Arbor Section of the International Association for Dental Research and the First District Dental Society of the State of Michigan, of Detroit. Held at the Michigan Union in Ann Arbor, May 10, 1928. Chairman: P. J. O'Reily, Detroit. There were afternoon and evening sessions, and a banquet between.

Scientific program

1. Afternoon session

I. Toxic substances (split-protein products) associated with periapical infections. *U. G. Rickert*, A.M., D.D.S. 75

II. Radium therapy. *Ernest A. Pohle*, M.D., Ph.D 76

2. Evening session

III. Finding lost radium. *Arthur W. Smith*, Ph.D 77

Abstracts

I. TOXIC SUBSTANCES (SPLIT-PROTEIN PRODUCTS) ASSOCIATED WITH PERIAPICAL INFECTIONS

U. G. Rickert, A.M., D.D.S., University of Michigan

The author called attention to the substances resulting from decomposition of the organic matter of dentine following pulp extirpation, and also of the decomposition of circulatory elements from the general circulation that diffuse through the more permeable end of a tooth after pulp extirpation, which may become toxic by stasis even in the absence of infection. Laboratory and clinical observations were presented indicating that such toxicity frequently precedes infection itself; that such traumatized teeth often become infected; that radiographic periapical changes take place following pulp extirpation in from two to six months even in the absence of infection; and that mechanical trauma hastens the development of such changes.

The work suggested that root surgery, in order to be effective, must inhibit diffusion through the root beyond the tolerance of the enveloping tissues; and that bacteria in the process of periapical infection, though highly important, may really only be incidents in the pulpless tooth problem. The methods used in root surgery at the University of Michigan to prevent diffusion through the root-end, with the clinical results, were also presented.

The guests were taken through the laboratory, where demonstrations of the subject matter were studied directly.

II. RADIUM THERAPY

Ernest A. Pohle, M.D., Ph.D., Professor of Radiology, University of Wisconsin

Radium is being used therapeutically in three forms: namely, (a) solid salt, (b) gas (emanation), and (c) solution (per os or by intravenous injection).

There are three available salts: the bromide, which contains 53.6 per cent of radium element; the sulphate, which contains 70.2 per cent; and the chloride, which contains 76.1 per cent. These salts are placed in needles or tubes, or can be spread over a flat surface. The latter type of application is called radium plaque. The emanation in gas form, called radon, is pumped off of radium salt in solution. The apparatus in which this process is carried out is called an emanation plant. While radium salt disintegrates to one-half its original strength in 1730 years, the radon loses about one-half its original strength in 3.85 days. If all the emanation is pumped off of a certain amount of radium in solution, it will be replaced in 60 days and may be pumped off again. The strength of the solid salt is expressed in milligrams of the element; the strength of the emanation, in millicuries. Radon is usually transferred to small containers made of glass, gold, or platinum, which, called implants, are implanted into tumors.

The radiation emitted by radium that interests us in therapy consists of alpha, beta, and gamma rays. The alpha rays are absorbed by a layer of cardboard or rubber, and most of the beta rays are absorbed by a 0.3 mm. layer of platinum. This leaves, then, only the very penetrating gamma rays, which have a short wave-length, in the

neighborhood of 0.01 angstrom. Superficial lesions are often treated by unfiltered radiation, while for deep seated malignancies filtration is necessary. If radon implants are inserted into a tumor, gold or platinum filtration prevents excessive necrosis. For dentists it is important to remember that early cases of carcinoma of the mouth, in the buccal mucosa, tongue, or palate, respond fairly well to radium treatment. It is their duty, then, to watch in each patient for suspicious lesions in the mouth, and to refer such cases at the earliest date to the proper place for treatment.

III. FINDING LOST RADIUM

Arthur W. Smith, Ph.D., Professor of Physics, University of Michigan

According to modern ideas every atom is a complicated structure, consisting of many smaller parts. Some kinds of atoms, of which radium is a good example, are continuously disintegrating by expelling some of these particles. There are three kinds of radiations from these atoms. The alpha particles are as massive as the atoms of helium, and are stopped by a very thin sheet of paper. The beta rays consist of particles that are 7200 times lighter, being high-speed electrons that require a thickness of 2 mm. of aluminum to stop them. Accompanying these disturbances in the atom are the gamma rays, similar to light or x-rays, but so penetrating that several inches of lead are required to stop them completely. When gamma rays pass through air they break up some of the molecules into ions. Half of these ions are electrically charged positively, the other half negatively. This makes the air slightly conducting for electric currents, and it gives a means for measuring the intensity of the gamma rays.

At this point in the lecture it was announced that the tube of radium that had been shown to the audience by the preceding speaker, during the banquet, had been left on one of the dishes and carried to the kitchen. The trays of dishes were then returned and carried near an electroscope, which had been brought in for exhibition. This electroscope consisted of a metal chamber enclosing about 15 liters of air. In the middle of the chamber was an electrically insulated rod charged to 500 volts. When gamma rays passed through the air in this chamber, the air became enough of a conductor to allow the charge on

the rod to leak away. The stronger the rays, the faster the charge disappeared. The charge was measured by a strip of gold leaf attached to the upper end of the insulated rod and observed through a microscope. The nearer radium was placed to this electroscope, the faster the image of the gold leaf was seen to move.

Tray after tray of the dishes was brought near the electroscope with no effect, until one tray caused the leaf to fall. Dish after dish from this tray was then placed near the electroscope until one dish caused the leaf to fall again. In melted ice cream in this dish was found the small tube of radium.

The same electroscope was used on several occasions to find radium that had been lost in different hospitals in the State. When a tube of radium has been through a furnace, the ashes are brought, pailful by pailful, to the electroscope until it shows that the radium is near. When the lost tube of radium is somewhere in the building, the electroscope is set up in one place after another until there is some indication of the presence of radium. Then, as in the old game of "hot and cold," the electroscope is moved nearer and nearer in the direction that gives the faster fall to the gold leaf.

INTERNATIONAL ASSOCIATION FOR DENTAL RESEARCH

Scientific Proceedings of the Chicago Section[1]

January 14, 1929

B. ORBAN, M.D., *Editor*

Meeting held at the West Side Student Y. M. C. A. Building, Chicago, January 14, 1929. Chairman: Edward H. Hatton, B.L., M.D., President of the Section.

Scientific program

I. The morphology and growth of the fusiform microörganisms of the mouth. *Theodore B. Beust*, M.D., D.D.S..... 79
II. The classification of tumors. *Carl Waldron*, M.D., D.D.S., and *Harold G. Worman*, D.D.S..... 80

Abstracts

I. THE MORPHOLOGY AND GROWTH OF THE FUSIFORM MICROÖRGANISMS OF THE MOUTH

Theodore B. Beust, M.D., D.D.S., School of Dentistry, Louisville, Ky.

The paper dwelt on the polymorphic microbial forms in the mouth. One representative of these organisms, the Leptothrix falciformis, first described by the essayist in 1908, was declared to be the parent of various fusiform elements in the mouth. These fusiform organisms were shown to exist, often in great numbers, in the oral cavity of all subjects, irrespective of the occurrence of lesions. The inference therefore appeared justified that the microscope was of little avail in the diagnosis of Vincent's angina. The paper was illustrated by a number of lantern slides and microscopic specimens.

[1] The last preceding series of abstracts of the proceedings of the Chicago Section was published in the *Journal of Dental Research*, 1928, viii, p. 613.

II. THE CLASSIFICATION OF TUMORS

Carl Waldron, M.D., D.D.S., and Harold G. Worman, D.D.S.,
Minneapolis, Minnesota

With all due respect for present classifications of tumors of the oral cavity, and tumors in general, the authors have felt the need of a "clinico-pathological" classification of tumors of the mouth and jaws. Such a classification should be easily understood and used by the general practitioner of dentistry, and should enable him to detect abnormalities earlier, diagnose them with a greater degree of certainty, and effect removals and cures of tumors without the extensive loss of normal healthy tissue or even life itself that sometimes occurs.

With regard to the odontomes, the British Dental Association's classification cannot at this time be improved upon. Furthermore, in the case of jaw tumors, the Codman Registry classification of tumors of bones in general is well adapted for use in this special field. But in the case of other oral tumors, such as those of the gingivae (epulides), for example, available classifications are more or less vague. They have varied from histological classifications on one hand to clinical and topographical classifications on the other, with all sorts of combinations between. The result is chaos, at least in the mind of the general practitioner. Too often we see the effects of surgical interference with a carcinoma or sarcoma by some well meaning dentist who thought he was dealing with an ordinary fibrous epulis.

With the above indication of the motive prompting the arrangement and definition of oral tumors, the reader is referred to Bunting's text-book on oral pathology,[2] which contains the new clinico-pathological classification.

[2] Waldron: Bunting's Text-book of oral pathology. 1929, pp. 420–89. (Lea and Febiger, Philadelphia.)

PAINFUL SOCKETS AFTER EXTRACTIONS[1]

A Preliminary Report on the Investigation of Their Etiology, Prevention, and Treatment

JOSEPH SCHROFF, M.D., D.D.S., and HENRY A. BARTELS, B.S., D.D.S.

Department of Oral Pathology, School of Dental and Oral Surgery, Columbia University, New York City

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I. INTRODUCTION

The control of pain, during the period of extraction of teeth and after their removal, is of vital importance to the dental profession. Through the proper administration of anesthetics, local or general as indicated, the dental practitioner possesses a method whereby he may eliminate pain during extraction. Post-extraction pain, however, cannot be so easily controlled since at present its etiology is not definitely known, and therefore no specific treatment can be advised. Unfortunately, in the past, more attention has been devoted to after treatment than to search for causes of the post-extraction pain and its prevention. Strange to say, the patient, through some peculiar reasoning, expects freedom from pain after an extraction, although he fully anticipates pain as a consequence of trauma or of surgery in any other part of the body. We all have observed that in many cases there is no after pain, while in other cases the after pain is very severe. Painful sockets are embarrassing to the dentist, since the patient

[1] Read at a meeting of the William Jarvie Society for Dental Research, Dental School of Columbia University, February 7, 1929.

believes the pain is the result of some flaw in the practitioner's technique.

The senior author, while a dental student, was attracted to this problem of painful sockets subsequent to extraction, and like many dentists wondered why there should be pain in some cases and not in others. When various authorities were questioned, they invariably answered that it was due to the novocaine used for the anesthesia. This idea has been so well drilled into the minds of dentists that it has been transmitted by them to the laity, who also now-a-days believe that there is more pain following the extraction of teeth with the aid of novocaine than with other anesthetics. Indeed, for this reason, some patients refuse to allow the use of novocaine.

It soon became obvious to us, however, that novocaine was not the all important factor in producing after-pain, because painful sockets were observed in individuals whose teeth were extracted under such general anesthetics as gas and oxygen, ether, etc. This fact was substantiated by dentists having extended experience in extracting teeth under "gas" in the days before novocaine was introduced. That novocaine when misapplied may produce local pain in the soft tissues is readily admitted, but these cases are not the true painful sockets. The unsterile needle, the too forceful injection of the solution into the taut gum around the tooth, etc., as causes of local inflammation, are self evident.

As our clinical experience widened, we observed that in certain types of cases painful sockets were apt to occur. Such cases were these:

(a) After difficult extractions of cuspids in otherwise edentulous mouths, in which there had been a condensation of alveolar bone about these teeth, owing to possible fracture of the labial plate.

(b) After extraction of teeth with hypercementosed roots. The post-extraction pain was thought to be due to the trauma induced in extraction through the "burnishing action" of the hypercementosed root against the alveolar bone.

In these two types of cases it was noticed that there was apt to be either a slight amount of bleeding, or that the blood clot did not remain fixed to the alveolar bone. The pain was considered to be due to inflammation resulting from irritation of the exposed bone by bacteria

from the saliva, or to the fact that the bone had been infected before the tooth was extracted (osteitis or local osteomyelitis). The idea that in these cases the pain was dependent upon exposure of bone was soon rejected. After difficult extractions, in which the use of a mallet and chisel was necessary, after-pain was comparatively slight or in some cases entirely absent. It was further observed that often no pain was evidenced when even relatively large areas of bone were exposed to the bacterial flora of the saliva.

The question then arose whether the toxins, or by-products of the enzymic activity of certain bacteria, are responsible for the severe after-pain. It is now assumed that the pain in inflammation is due to action of the toxins and by-products of bacteria upon nerve endings rather than to pressure. To which group of microörganisms could this action be most logically attributed? From our clinical experience with early cases of Vincent's infection, our attention was directed to these anerobic organisms, since one of the early characteristic symptoms in a true Vincent infection is tenderness or pain about the teeth to a far greater degree than the lesions warrant. With this thought in mind we decided to check up bacteriologically all the painful sockets we encountered after extractions, to see what our findings would be and what conclusions if any could be drawn therefrom.

II. CLINICAL AND BACTERIOLOGICAL FINDINGS

Case 1. Patient G. D.; male, white, age 35. *Sept. 20, 1926.* Treated for Vincent's infection. *Nov. 15.* Returned for extraction of lower, left, second bicuspid which was malplaced. Difficulty was encountered owing to position of root. *Nov. 17.* Patient complained of pain in socket and also about mouth. Smears of socket and mouth showed numerous fusiform bacilli and spirochetes. Treatment consisted of irrigation of socket, and use of sodium perborate and parathesin. *Nov. 24.* Patient returned after having been out of town for a week. Socket was irrigated, and pocket distal to third molar cleaned out. Sodium perborate was continued. *Nov. 26.* Patient reported sufficiently improved to warrant scaling of teeth where necessary. Sodium perborate was continued. *Nov. 28.* Patient discharged. Advised to continue sodium perborate for several weeks.

Case 2. Patient G. R.; male, white, age 24. *Jan. 6, 1927.* Complained of severe pain in sockets of upper right and upper left second bicuspids, which had been extracted on *Jan. 3* by a dentist in Pittsburgh. Patient

states he was unable to sleep for three nights owing to severe pain. *Physical examination.*—Patient apparently in good physical condition. Remaining teeth appeared to be normal. Sockets were dry, and contained no blood clot; bare bone could be seen. *Treatment.*—Irrigated with hot saline. Parathesin powder and iodoform gauze were placed in the sockets. Codeine sulphate and pyramidon were also prescribed. *Jan. 7.* Pain had subsided considerably overnight. Irrigated and re-dressed, and benzoin and parathesin powder used. *Jan. 8.* Re-irrigated and re-dressed. *Jan. 12.* Patient complained of pain about upper cuspid teeth. Gums were bleeding slightly in this area. Patient stated that he had same type of pain prior to extractions in Pittsburgh. Further questioning of patient elicited information that there were no cavities in teeth extracted. Examination revealed two slight ulcers covered with greyish slough, each about 1 mm. in diameter, one lying on alveolar ridge of lower left jaw on mesial side of second molar; the other, on mesial side of upper second molar. These areas bled easily and were very tender. Smears showed numerous fusiform bacilli and spirochetes. Sodium perborate was administered. *Jan. 22.* Patient discharged, pain having subsided.

Case 3. (History through the courtesy of Dr. M. Appel, Bayonne, N. J.) Patient A. J.; male, white, age 30. Mouth clean and teeth in good condition with the exception of upper, right, first molar, which showed evidence of infection. *Mar. 18, 1927.* Upper right first molar extracted, using novocain. Socket dressed with iodoform gauze and parathesin. *Mar. 19.* Considerable bleeding occurred on removal of gauze. Socket irrigated with warm saline. *Mar. 23.* Tenderness on palatal margin of socket, patient unable to eat. Pain in all teeth, upper and lower, on right side. Bleeding from socket accompanied by fetor of breath. Socket irrigated with hot saline. *Mar. 24.* Condition not improved. Smears contained fusiform bacilli and spirochetes. Socket irrigated with concentrated sodium perborate, and patient advised to use perborate as mouth wash. *Mar. 25.* Condition much improved. Continued perborate treatment. *Mar. 26.* Condition much improved. *Mar. 28.* Socket still open, no pain. *April 4.* Pus flowing from socket; removed several pieces of loose bone. *April 8.* Socket healing normally.

Case 4. (History through the courtesy of Dr. M. Appel, Bayonne, N. J.) Patient C. B.; male, white, age 23. *May 18, 1927.* For two weeks, mouth and teeth very sore; not able to eat properly. Gums about all teeth had characteristic lesions, and breath a fetor symptomatic of Vincent's infection. Teeth in poor condition, having heavy deposits and many were carious. Case treated with sodium perborate. *May 20.* Condition not much

improved. Sodium perborate continued. *May 21.* Condition much improved; not much pain or fetor of breath. Sodium perborate continued. *May 25.* Condition improved sufficiently to permit scaling and cleaning of teeth. *May 27.* Extracted lower, left, second molar, using novocaine. *May 28.* Socket very painful; bleeding and fetor of breath. Patient unable to work for several days after extraction. Irrigated with a concentrated solution of sodium perborate, and patient continued using same as mouth wash. *June 1.* Condition improved, but still complaining of pain from side of mouth where tooth was extracted. Sodium perborate continued. *June 2.* Condition improving. Sodium perborate continued. *June 10.* No pain, no active Vincent's lesions. Gums had normal pink color and were tight around teeth.

Case 5. Patient G. L. B.; male, white, age 24. *Dec. 17, 1927.* Appeared for extraction of lower, right, second molar root; mouth was in fairly good condition. Sodium perborate powder was placed in socket following extraction. *Dec. 18.* No pain following extraction. *Dec. 21.* Patient had dull pain in socket; stopped using perborate (against instructions) on Dec. 18. Slight greyish membrane about socket. Smear showed numerous fusiform bacilli and spirochetes, few cocci, and some leukocytes. Socket irrigated with hot sodium perborate. Patient instructed to swab area with sodium perborate every hour. *Dec. 24.* Patient reported pain had disappeared. Smear from socket contained no spirochetes or fusiform bacilli.

Case 6. Patient V. L. L.; female, white, age 30. Radiographic examination revealed radiolucent areas about distal root apex of lower, right, first molar, and lower, right, first bicuspid. *Dec. 29, 1927.* Extracted these teeth, mandibular injection given, and areas about roots curretted. Owing to excessive bleeding, sockets were packed with iodoform gauze and aristole powder. *Dec. 31.* Patient complained of pain under tongue. Examination revealed hemotoma. Patient said teeth had been extracted during menstrual period. Hot saline irrigations at half-hourly intervals advised. *Jan. 1, 1928.* Hemotoma subsiding, irrigations continued. *Jan. 2.* Hemotoma practically gone, patient now complaining of pain in sockets. Smears contained fusiform bacilli and spirochetes. Sockets irrigated with hot sodium perborate and patient advised to apply sodium perborate to sockets at hourly intervals. *Jan. 4.* Socket reirrigated and lightly dressed to prevent entrance of food. *Jan. 6.* Pain had disappeared. Smears contained neither spirochetes nor fusiform bacilli.

Case 7. Patient A. W.; male, white, age 31. *Feb. 4, 1928.* Patient had lower, left, third molar removed at a clinic. *Feb. 6.* Patient complaining of pain in region of this socket. Smears contained fusiform bacilli

and spirochetes together with cocci. Sodium perborate treatment started as outlined above. *Feb. 10.* Pain had subsided. Smears of socket contained neither fusiform bacilli nor spirochetes.

Case 8. Patient K. B.; female, white, age 25. *Mar. 14, 1928.* Upper, left, third molar extracted, owing to caries. *Mar. 15.* Patient complained of pain. Irrigated with saline. *Mar. 16.* Patient still complained of pain. Smears of socket contained many fusiform bacilli and spirochetes. Sodium perborate treatment used as prescribed in former cases. *Mar. 18.* Patient stated pain had subsided considerably. Treatment continued. *Mar. 21.* Patient stated pain had disappeared. Smears contained neither fusiform bacilli nor spirochetes.

III. DISCUSSION

Although the finding of fusiform bacilli and spirochetes in the cases of painful sockets cited above is suggestive, it does not warrant the conclusion that they incited the condition observed. Fusiform bacilli and spirochetes are present in both normal and diseased mouths. Keilty (1), in a bacteriological study of the gums in 200 cases in which the mouth was suspected of being a focus of infection, found that in 69 percent either one or both of these organisms were present. Clinically the majority of his cases had a gingivitis of some type. Burns (2), in examinations of the mouths of 120 mothers with receding gums, found that these organisms were present in 80 percent of these patients. Miller and Epstein (3), in an extensive investigation of the significance of fusiform bacilli and spirillae in lesions of the mucous membrane, observed, in their study of smears from the gums of 160 individuals with normal mouths, that spirillae were present in 77 percent and fusiform bacilli in 92.5 percent of the total number. Pratt (4), in an examination of over 200 mouths, 50 of which were diagnosed as normal, found that fusiform bacilli were present in each. Davis (5), maintains that fusiform bacilli and spirochetes belong to the group "opportunists;" that is to say, they lead a more or less saphrophytic life, but still are potentially pathogenic. The trauma incidental to any extraction may produce a *locus minoris resistentiae*, thus affording an ideal area in which these bacteria may grasp their opportunity to proliferate and assume their pathogenic rôle. Attention is called to the fact that in several cases cited above (histories 1, 2 and 4), the patient at one time or another was suffering from Vincent's infection.

In all the cases controlled by smear examination, it was noted that fusiform bacilli and spirochetes were present in the painful sockets; and that upon disappearance of pain, presence of the organisms was no longer demonstrable.

In view of these findings, it was decided to attempt to inhibit the growth of these microörganisms by rendering the environment unsuitable for their development. Since anerobes find any area containing an abundance of oxygen deleterious to their growth, sodium perborate was employed as a bacteriostatic agent. We had the patient rinse his mouth with a solution of perborate before the tooth was extracted. After extraction, the socket was irrigated with hot sodium perborate solution, and the patient then dismissed with instructions to use it as a mouth wash as frequently as possible, preferably every half hour. No anodyne drug was prescribed, since we wished to exclude all extraneous factors that might influence our results.

In about 400 cases of extraction in private patients, in which the above procedure was followed, post-extraction pain of some degree occurred in about 5 per cent of all individuals so treated. When after-pain occurred, we noticed that it was generally experienced by patients in which the mouth of the socket had occluded. The use of a hot sodium perborate irrigation reaching the base of the socket invariably produced instantaneous relief. By keeping the mouth of such sockets open, to allow ready access of the sodium perborate, the sockets soon were rendered painless. In several instances, after-pain was found to be due to fracture of the process.

The importance of the anerobic streptococci as a possible factor in the production of after pain may not be ignored in the light of the recent investigations of Dr. Harold Neuhoff (6) of the Mount Sinai Hospital, New York City, upon the rôle of these organisms. He found them in lung abscesses and other metastatic conditions, and considers them the main incitant of these disturbances. However, the treatment prescribed above is efficacious against all anerobes, including this group of streptococci.

IV. SUMMARY

Fusiform bacilli and spirochetes have been found in cases of painful sockets following extractions.

Upon the cessation of pain, these organisms were no longer observed in smears of exudate from the sockets.

The use of sodium perborate as an aid in prevention and treatment of painful sockets is advised.

V. CONCLUSION

Our findings, clinical and bacteriological, indicate that anerobic organisms produce more painful sockets following extraction than do aerobes. Investigation along these lines is being continued by both aerobic and anerobic methods.

In order to compile data covering a greater series of cases, the writers desire to obtain case histories of painful sockets, accompanied by smears or swabs containing exudate from the sockets at the time the patients complain of this condition; also after the sockets are again painless. The histories and smears should be forwarded to the Department of Oral Pathology, School of Dental and Oral Surgery, Columbia University, New York City. Credit will be given each dentist submitting such material.

VI. REFERENCES TO LITERATURE

(1) KEILTY, R. A. 1922 Focal infection and bacteriological study of the gums in 200 cases. *Journal of Medical Research*, xliii, p. 377.

(2) BURNS, E. M. 1926 Vincent's infection. Significance as precurser of pyorrhea, and its possibilities as cause of other diseases. *California and Western Medicine*, xxv, p. 344.

(3) MILLER, H. E., AND EPSTEIN, N. 1926 The significance of fusiform bacilli and spirilla in mucous membrane lesions. *Ibid.*, xxiv, p. 633.

(4) PRATT, J. S. 1927 On the biology of B. fusiformis. *Journal of Infectious Diseases*, xli, p. 461.

(5) DAVIS, D. J. 1926 Pathology and bacteriology of putrid and gangrenous processes with special reference to fusiform bacilli and Vincent's spirochete. *Journal of the Iowa Medical Society*, xvi, p. 76.

(6) NEUHOFF, H. 1929 Personal communication.

MEDICO-DENTAL CASE RECORDS: SEVENTH REPORT[1]

A Clinico-Pathological Study

BISSELL B. PALMER, Jr., D.D.S., F.A.C.D., and MALCOLM W. CARR, D.D.S.[2]

New York City

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I. Introduction

It is now generally recognized that a chronic focus of infection may be the dominant etiologic factor in any one of a large group of systemic diseases. These lesions may be varied and situated in almost any part of the body. In the study of chronic disease of focal origin, it is not unusual to observe the coexistence of more than one secondary lesion in the same patient. Thus, by metastasis from the same primary focus, *multiple* secondary lesions may be produced and may exist concomitantly in the elementary tissues of the body as well as in any of the organs. Although such multiple secondary lesions appear as separate pathologic processes, the various symptoms of which are

[1] The previous reports were published in the *Journal of Dental Research:* (1) 1926, VI, p. 283; (2–4) 1927, VII, pp. 115, 275, 457; (5–6) 1928, VIII, pp. 73, 579.

[2] Presented at a session of the Seventh Annual Meeting of the International Association for Dental Research, Northwestern University Dental School, Chicago, Ill., March 23–24, 1929.

characterized by the nature of the tissue invaded, the primary focus of infection may be the original common etiologic factor. This report is the first in our series of medico-dental case records to present this phase of focal infection in its relation to chronic disease.

The patient described in this report presented three distinct *groups* of symptoms. This complexity of pathologic processes becomes apparent as the varied subjective symptoms are described in the medical history, and as further symptoms are revealed by the physical examination and clinical pathology. Lassitude, malaise, and weakness are the first symptoms described, and characterize a general state of lowered resistance and chronic invalidism that so frequently result from focal sepsis. The symptoms of generalized muscular pain and stiffness, and later the localization of pain in certain muscles and joints and the associated swelling of the joints, are the second manifestation of metastatic infection and indicate an involvement of the joints and fibrous tissue. Finally, invasion of the tissues of the cardio-vascular-renal system becomes apparent from the symptoms of dyspnea (and later the alarming collapse), the hypotension, and the albuminuria. The correlation of these varied symptoms, with the findings of the physical examination and the laboratory analyses, established the diagnosis of the two separate pathologic processes.

II. Case A-10 (1)[3]: Polyarthritis and Rheumatic Carditis (2)

General History

A married woman, forty-seven years of age, was admitted to the hospital[4] from the out-patient dispensary on May 5, 1928. Her chief complaint (3), at the time of admission, was generalized muscular and joint pain, particularly in the back and left chest.

Family history (4). Her father died at the age of forty-five of rheumatic fever; her mother died of an unknown cause; her husband and three children were living and well. The family history was otherwise negative.

Habits (5). She drank alcoholic beverages occasionally, but had never used tobacco. House work was her occupation.

Past medical history (6). She gave no history of scarlet fever,

[3] Numerals in parenthesis refer to numbered items in "Notes and comment," page 99.
[4] The Fifth Avenue Hospital, New York City.

diphtheria, typhoid fever, chorea, or venereal disease. She had rheumatic fever at fifteen years of age, with recurring attacks at three-year intervals for a period of twenty years. Then there was a period of remission until nine years ago, when she had an acute attack with polyarthritis. For the past few years she has had intermittent "heart attacks," and occasionally suffers from asthma at night.

History of the present illness (7). In June, 1927, one year before admission to the hospital, the patient developed a constant feeling of lassitude, malaise (8), and indisposition to work. At times there would be an overwhelming feeling of weakness. On June 18 she suffered from an attack of acute arthritis, involving the joints of the shoulders, ankles, and wrists. There was stiffness of the neck, and intercostal and lumbar pain, but no swelling of the joints. During this attack she had sweats and chills, fever and dyspnea (9). There was no cough (10). She lost her appetite, slept poorly, and lost weight. On July 1, she was seen in the out-patient dispensary and complained chiefly of pain in the shoulders. Physical examination revealed chronic endocarditis (11), myocarditis (12), slight cardiac hypertrophy (13), and polyarthritis. The pulse rate was 90; temperature, 99.4. She was given tr. digitalis (14) m. *xx* t.i.d., and sodium salicylate (15) gr. *xx* t.i.d., and ordered to rest in bed. During the interlude between July 1 and her next visit to the out-patient dispensary on November 29, she had two attacks of acute illness, during the first of which there was swelling of the joints, particularly the hands and knees, accompanied by fever. The condition improved with rest in bed and administration of salicylates. Late one night, about two months after this attack, she was attended at home by her physician. She was found to be in collapse. The body surface was cold and clammy; the respirations were orthopneic (16); the pulse rate was 140 and of poor quality. Her condition was again improved slowly by rest in bed and administration of digitan (17). When seen in the dispensary on November 29, P_2 (18) was louder over the aortic area (19) than over the pulmonic area (20). There was a loud systolic murmur (21), but no presystolic murmur (22) was audible.[5] The pulse rate was 86. One digitan tablet every other day was

[5] There evidently had been a presystolic murmur at one of the previous examinations but it was not recorded in the history.

prescribed. She came to the dispensary for observation at irregular intervals from December 1927 to March 1928. During this period the administration of digitan was continued. There had been an improvement in her cardiac condition although the joint pains persisted. In March, the pain seemed to be most severe in the feet, particularly the left foot, and she was referred to the orthopedic department for possible foot strain. It was concluded, however, that "the pain in the feet was probably a manifestation of the same arthritic process which was acute about six months ago when the hands and knees were involved."

There was no interval history from March until May, when the patient was admitted to the hospital for observation and diagnosis. When she entered the hospital her chief complaint was muscular and joint pain associated with stiffness. Many other symptoms were also being manifested. There was weakness, fever, swelling of the ankles, and dyspnea. The appetite was poor, the bowels were constipated, and she slept irregularly. She had again lost weight. Her best weight had been 140; at the time of admission it was 135.

PHYSICAL EXAMINATION

Examination showed a fairly well developed but poorly nourished woman. She appeared chronically ill.

Eyes. There was slight sclerosis of the retinal arteries (23). The reaction to light and accommodation was normal (24).

Mouth and teeth. Clinical examination.—There were only six anterior teeth in the mandible (the left central incisor to the right second bicuspid inclusive), and seven in the maxilla. The left maxillary second molar was the posterior abutment for a bridge, the anterior abutment being the lateral incisor. Both abutments were gold-shell crowns. The left central incisor remained. On the right side the lateral incisor supported an extension, anteriorly, to supply the missing central incisor, and served also as the anterior abutment of a bridge to the first bicuspid, supplying the missing cuspid. The first bicuspid had an extension to supply the missing second bicuspid. The first and second molars remained. There was a heavy deposit of salivary calculus and an advanced chronic suppurative gingivitis (25). In the region of the mandibular anterior teeth there was con-

siderable resorption of the alveolar process, with retraction of the soft tissues at the gingival margins, resulting in the formation of deep pyorrheal pockets of infection, from which an exudation of free pus could be expressed. This same condition was found at the gingival margin of the right maxillary lateral incisor and second bicuspid. There was considerable gingival irritation from the bridge on the right side, which was unsanitary and poorly fitting. Oral hygiene had obviously been neglected. *Roentgenographic examination.*—Roentgenograms showed extensive alveolar resorption in the region of the remaining mandibular teeth. There was no evidence of periapical (26) infection of the left maxillary second molar, lateral or central incisors. The right lateral incisor was a pulpless tooth and showed chronic rarefying osteitis (27) at the apex. There was also severe destruction of the periodontal (28) tissues. The right first bicuspid remained in the center of a necrotic area. There had been complete destruction of the investing alveolar process to and beyond the apex, and for a considerable distance anteriorly and posteriorly. The right first and second molars showed no evidence of periodontal or periapical infection. It was believed that the oral foci of infection were probably the dominant etiologic factor (29) in the patient's general condition; and it was recommended that the remaining mandibular teeth, the septic bridge on the left side of the maxilla, and the right maxillary lateral incisor and first bicuspid, be removed.

Naso-pharynx. The nasal septum was moderately deviated to the right; the breathing space was somewhat reduced; the mucous membrane appeared normal.

Tonsils. The tonsils were small, irregular, and not markedly diseased. Small amount of caseous debris was expressed from the left tonsil. The anterior cervical lymph-nodes were negative.

Sinuses. No tenderness was elicited. The right maxillary sinus transilluminated slightly darker than the left (30).

Lungs. There were medium moist râles (31) at the bases. Resonance (32) was diminished at the right base. Roentgenographic examination of the chest showed a slight congestion at the right base.

Heart. There was a loud systolic murmur; P_2 was louder than A_2 (33), but the accentuation was not great. Mensuration of the heart by means of percussion upon the chest wall indicated slight enlarge-

ment. Roentgenographic film of the chest at a distance of six feet showed an enlargement of the heart and confirmed the diagnosis made by percussion. The enlargement was in the region of the left auricle (13). The transverse diameter of the heart was 6 inches and that of the chest 10¾ inches (34). *Report on the electrocardiogram* (35).—"Normal sinus mechanism (36); average recorded rate 80. Time relationships: the P-R time (37) reaches the maximal normal limits of 0.20 second in lead II (38). This is either the toxic effect of infection or digitalis. General conformation: moderate depression of T-waves in all leads, especially I and II. This also suggests a digitalis effect; but if the patient is not digitalized, it indicates myocardial damage. No preponderance." *Comment of the cardiologist.*—"The cardiac condition appears quiescent and the impression received from the physical examination is that an active carditis is not now present, although its presence should be assumed in all recurring attacks of rheumatic fever. Mitral insufficiency (39) appears to be the chief valvular lesion, as signs of stenosis (40) could not be elicited at the time of examination. The heart does not appear greatly enlarged. The chief problem is the elimination of focal infection from the mouth and upper respiratory tract (tonsils). I do not believe digitalis is indicated now, but that salicylates should be pushed, especially when foci of infection are being opened"

Abdomen. The liver (41) was palpable 3 cm. below the costal margin, and tenderness (42) was elicited upon palpation. There were no other regions of tenderness or rigidity (43), nor were there any masses (44).

Extremities. The ankles were slightly swollen and painful. Reflexes were normal.

CLINICAL PATHOLOGY (LABORATORY REPORTS)

For the first two days after admission to the hospital the patient's temperature ranged from 98.2 to 99.8; after the second day it remained within normal limits. The pulse rate ranged from 60 to 75. The rate of respiration was within normal limits.

Weight: 135 pounds.

Blood pressure (45): systolic 110 mm.; diastolic, 60 mm.

Urinalysis (46). Amount, normal; specific gravity, 1,020; reac-

tion, acid; albumin, faint trace; sugar, acetone, diacetic acid, absent. *Microscopic examination.*—Casts, absent; red blood cells, absent; white blood cells, few; epithelia, moderate number.

Hematology (47): hemoglobin, 87 per cent; red blood cells, 4,100,000; white blood cells, 12,000; polymorphonuclear leukocytes, 64 per cent; lymphocytes, 34 per cent; eosinophilic leukocytes, 2 per cent; non-lobulated neutrophilic leukocytes, 56 per cent.

Blood chemistry (48); mg. per 100 cc.: urea nitrogen, 15.8; uric acid, 2.1; sugar, 111; CO_2-combining power, 48 per cent (volume).

Blood complement fixation test (49); cholesterol-alcohol: negative.

Kahn test (50): negative.

Blood culture (51): no growth after seven days.

Kidney functional tests. Phenolsulphonephthalein test (52): first hour, 42 per cent; second hour, 13.6 per cent; total, 56.6 per cent.

Mosenthal test (53):

Time	*cc.*	*Spec. gr.*		
10 A.M.	400	1.016	Night chlorides	3.7 gm.
12 Noon	100	1.015	Day chlorides	5.2 gm.
2 P.M.	100	1.013	Total	8.9 gm.
4 P.M.	75	1.014		
6 P.M.	100	1.015		
8 P.M.	100	1.015		
Total	875			
Night	355	1.017		

DIAGNOSIS

A. Acute polyarthritis.

B. Subacute rheumatic carditis: mitral insufficiency with moderate cardiac hypertrophy.

C. Toxemia from oral sepsis.

TREATMENT AND OPERATIONS

Medical treatment. The patient received digitalis gr. *iss* once each day. Sodium salicylate gr. *xxx* and sodium bicarbonate gr. *xx* were given three times a day. Rest in bed was required and the diet was studied.

Oral surgical treatment. The infected teeth were removed in two

stages (54); 1 per cent procain, containing epinephrin 1–40,000, was the local anesthetic solution employed.

First operation, May 9. The right maxillary lateral incisor and first bicuspid were removed. Infection from the incisor had caused a chronic rarefying osteitis. There had also been considerable destruction of the alveolar process owing to periodontal disease. This destruction extended posteriorly to the first bicuspid region, where it expanded into an extensive necrotic area. The first bicuspid was in the center of this area of necrosis, and was surrounded by pathological granulation tissue. The areas of infection were curetted, cauterized, and kept open by the usual post-operative dressings until normal granulations had taken place.

Second operation, May 11. The six remaining mandibular teeth (left central incisor to the right second bicuspid) were removed. Advanced periodontal disease had produced an extensive resorption of the alveolar process, so that the teeth were retained by little more than soft tissue attachment. The wounds were curetted lightly, and dressed until they had healed normally. The advanced periodontal disease precluded the possibility of obtaining uncontaminated cultures. At a subsequent date the unsanitary bridge was removed, and special oral hygiene treatment was instituted in order to restore to health the diseased periodontal tissues surrounding the remaining maxillary teeth.

PROGRESS NOTES

There were *no* exacerbations (55) following the removal of the infected teeth and the consequent opening of the septic foci.

Clinical pathology. Urinalysis (May 15).—Reaction, acid; specific gravity, 1,020; albumin, negative; sugar, negative; casts, negative; red blood cells, negative; white blood cells, few. *Hematology* (*May 15*).—Hemoglobin 83 per cent; red blood cells, 4,200,000; white blood cells, 5,900; polymorphonuclear leukocytes, 60 per cent; lymphocytes, 37 per cent; mononuclear leucocytes, 2 per cent; eosinophilic leukocytes, 1 per cent; non-lobulated neutrophilic leukocytes, 58 per cent. On May 14 the patient was "symptom free," although the presystolic murmur was still present. On May 19 there had been no return of the rheumatic pains and the patient's condition was described as ex-

cellent. On May 20 the patient was discharged from the hospital "improved."

FOLLOW-UP EXAMINATION

The patient was seen for follow-up examination at three month intervals, in August and November, 1928, and February, 1929. The marked improvement in the subjective symptoms, and the physical examination findings at the first two follow-up examinations indicated that the patient had improved greatly in general health. She reported to the dispensary in February, nine months after discharge from the hospital, for the final examination, the findings of which are summarized in the following resume—all medication had been discontinued since the patient's discharge from the hospital in May.

Subjective symptomatology (56). There was either improvement in, or entire relief from, all the subjective symptoms which had been important during the course of the disease. There was relief from the general feeling of lassitude and there was now no weakness, no sweats nor chills, no fever. There had been an entire relief of all rheumatic pain and muscular stiffness. The ankles were no longer swollen, and dyspnea was much less prominent, being manifested only upon exertion. The appetite was improved. There was still constipation and she slept rather poorly.

Physical examination. The patient's general appearance had improved; she was now alert and the countenance was bright. Temperature, 97; pulse 64; respiration, 18; weight, 136 pounds; blood pressure, systolic, 134 mm., diastolic, 80 mm.

Teeth. The remaining maxillary teeth had been removed in order that a more satisfactory denture could be constructed. The mucous membrane was normal and the wounds were entirely healed.

Tonsils. The tonsils had not been removed. They were small and not inflamed. The throat appeared normal.

Chest: normal resonance; no râles.

Heart. The heart was moderately enlarged. The first sound at the apex was of fair quality and partially displaced by a systolic murmur. The presystolic murmur was not audible either in erect or recumbent posture. P_2 was greater than A_2. The pulse rate was 64, and the rhythm was regular.

Abdomen. The liver was enlarged three fingers breadth below the costal margin, but did not elicit tenderness. The spleen was not palpable, and there were no masses or areas of rigidity.

Extremities. There was no edema or swelling of the joints. Pain was absent. Reflexes were normal.

Clinical pathology. Urinalysis.—Reaction, alkaline; specific gravity, 1.001; albumin, negative; sugar, negative; casts, negative; red blood cells, negative; white blood cells, few. *Hematology.*—Hemoglobin, 100 per cent; red blood cells, 5,000,000; white blood cells, 9,000; polymorphonuclear leukocytes, 61 per cent; lymphocytes, 37 per cent; eosinophilic leukocytes, 1 per cent; transitionals, 1 per cent; non-lobulated neutrophilic leukocytes, 49 per cent. *Blood chemistry* (mg. per 100 cc.).—Urea nitrogen, 10; uric acid, 2.3; sugar, 87.7. In addition to the marked improvement of all the subjective symptoms, physical examination showed an increase in weight and a decided improvement in the blood-pressure. The urinalysis was now normal, whereas before there had been a slight albuminuria. Hematology showed an increase in both the hemoglobin and the number of red blood cells, which were now normal, and a decrease in the number of the white blood cells. The percentage of the non-lobulated neutrophils had become reduced and the blood chemistry was normal.

III. General Summary

The patient had a series of recurrent attacks of rheumatic fever in early life, followed by a period of remission with one exception, until she came to the dispensary at the age of forty-seven. The history of the present illness records three groups of symptoms; namely, (a) of general lowered resistance, (b) in joints and fibrous tissues, and (c) in the cardio-vascular-renal system. The symptoms at this time were not entirely characteristic of rheumatic fever, although in many respects resembling it. The cardiac condition improved with medical treatment and rest, but the arthritis and general lowered resistance persisted until the patient was admitted to the hospital for a complete study and observation, when extensive oral sepsis was revealed, and examination for foci elsewhere in the body was negative.

The oral foci were eliminated and the patient given routine general care. At the follow-up examination, nine months after the patient's

discharge from the hospital, there was complete relief from all of the symptoms in the joints, and a marked sustained improvement in all subjective symptoms.

IV. Conclusions

(1) Multiple secondary lesions may be produced by metastasis from the same primary focus, and may exist concomitantly in various tissues of the body.

(2) Symptoms of secondary lesions of focal infective origin probably do not show sustained improvement under medical treatment so long as the primary focus remains active.

(3) Oral sepsis may be the *dominant etiologic* factor in the production of lesions of the joints, muscles, and fibrous tissues, as well as the tissues of the cardio-vascular-renal system.

(4) Oral sepsis may be an aggravating or contributing factor to emphasize the symptoms of lesions produced originally by rheumatic fever.

V. Notes and Comment

Explanatory and Supplementary

(1) Case A-10. The cases reported in this series are classified according to the diseases that are being studied in relation to focal infection. Thus, the serial number A-10 indicates that this patient was the tenth case to be studied in the *arthritis* group.

(2) *Polyarthritis:* simultaneous inflammation of several joints. *Rheumatic carditis.*—The inflammatory affection of the several tissues of the heart caused by rheumatic fever. (Sturges suggested the term *carditis* because in many cases the endocardium, myocardium, and pericardium all become affected.) Inflammation of the cardiac tissues, irregular fever, and polyarthritis, are characteristic of rheumatic fever. Recurrent attacks of this affection are characteristic of the disease, and not only does an attack not confer immunity, but as in pneumonia, predisposes the subject to the disease.

(3) Chief complaint. The chief complaint is the expression by the patient of the symptoms that cause greatest distress, and which immediately draw attention to the site of disease and indicate the line of investigation to be followed.

(4) Family history. Enquiry into the family history may reveal certain

hereditary tendencies, and is important in its relation to predisposition to disease.

(5) Habits. The patient's habits and personal hygiene may be predisposing factors to disease.

(6) Past history. Enquiry into the past medical history is made for the purpose of ascertaining the previous diseases that may have a bearing upon the present illness. In this case the past history of rheumatic fever is an important item.

(7) History of the present illness. In the compilation of a complete medical history, a detailed sequential "history of the present illness" is one of the most essential parts of the record, and should contain all the subjective symptoms described by the patient.

(8) Lassitude and malaise: symptoms that most frequently accompany a low-grade toxemia and lowered resistance.

(9) Dyspnea. The symptom of dyspnea in this case refers to a shortness of breath rather than to difficult or labored breathing. Dyspnea is a symptom of cardio-vascular disease.

(10) Cough. The symptoms of loss of weight, fever and sweats, as described in this history, could relate to pulmonary tuberculosis as a complication. Cough, being also an important symptom of pulmonary tuberculosis, is noted here and indicates the historian's trend of thought.

(11) Endocarditis: inflammation of the endocardium or lining membrane of the heart, associated with valvular lesions.

(12) Myocarditis: inflammation of the myocardium or heart muscle, a condition that frequently accompanies endocarditis.

(13) Cardiac hypertrophy: increase in the size of the heart. Overgrowth of the musculature of the heart is the result of over-work and inflammatory injury. An obstructive lesion between the left auricle and the left ventricle (mitral stenosis), produces enlargement of the left auricle. Cardiac hypertrophy is therefore usually compensatory.

(14) Digitalis: a drug used as a cardiac stimulant. It stimulates and strengthens the muscle fibers of the heart, thereby increasing pulse force and arterial tension. It tends to slow the pulse and regulate the rhythm.

(15) Sodium salicylate: the salt usually employed to secure the constitutional action of salicylic acid. The chief sphere of usefulness of sodium salicylate is in the treatment of acute rheumatic fever, in which it seems to have a specific action. Alkaline carbonates are usually prescribed with the salicyates, as they lessen the gastric action by preventing the formation of the irritant salicylic acid.

(16) Orthopnea: ability to breathe with comfort only when sitting erect or standing.

(17) Digitan: a digitalis compound.

(18) P_2. The symbol P_2 represents that portion of the second heart sound which is due to the sudden coaptation of the valves that guard the orifice of the pulmonary artery. This sound is best heard at the base of the heart in the second left interspace. Regurgitant and stenotic lesions of the mitral valve result in accentuation of the pulmonary sound (P_2).

(19) Aortic area: the area at which the sound produced by closure of the aortic valve is most clearly audible; situated in the second intercostal space immediately adjacent to the right sternal border.

(20) Pulmonic area: the area at which sounds generated by the action of the pulmonary valve are most distinctly audible; a point immediately to the left of the sternum in the second intercostal space.

(21) Systolic murmur: occurs during contraction of the ventricle (systole). A systolic murmur heard at the apex, and in the left axilla, is present in mitral insufficiency.

(22) Presystolic murmur: audible just prior to the first heart sound; usually associated with mitral stenosis.

(23) Sclerosis of retinal arteries: determined by ophthalmoscopic examination, suggestive of general arteriosclerosis.

(24) Pupillary reactions. (a) Reaction to light: the normal pupil, when exposed suddenly to light stimuli, responds by a reflex contraction of the iris. A sluggish reaction to light, or total abolition of the light reflex, signifies optic atrophy, partial or complete paralysis of the third cranial nerve, or degenerative changes in the ciliary ganglion. (b) Reaction to accommodation: when the range of vision is suddenly transferred from a distant objective point to an object near at hand, the pupils contract and the eyes converge. This is the reaction to accommodation. Abolition of this reflex is due to paralysis of the third cranial nerve.

(25) Chronic suppurative gingivitis: a disease process that induces a breaking down of the tissues supporting the teeth (periodontoclasia).

(26) Periapical: relating to the region of the alveolar process about the apices of the roots of the teeth.

(27) Chronic rarefying osteitis: a condition resulting from the thickening of the periodontal membrane that takes place at the expense of the bone of the alveolar process. As proliferation of the round cells occurs, the bone is rarefied and finally destroyed, leaving the space filled with chronic inflammatory tissue, the granuloma.

(28) Periodontal: relating to the supporting tissues surrounding the teeth.

(29) Etiologic factor. Following the physical examination and a complete study of the history of the case, an estimation is made as to whether the oral condition of the patient is probably an etiologic factor, or an aggravating factor, or not a factor in the patient's general condition.

(30) Difference in transillumination. The deviation of the nasal septum or the chronic right maxillary infection might have caused the right maxillary sinus to transilluminate darker than the left maxillary sinus.

(31) Râles: adventitious sounds generated in the pulmonary tract; caused by the pressure of serum, mucous, pus, or blood in the air passages, which interfere with the free ingress and egress of air.

(32) Resonance: the sound elicited on percussion of the surface of the thorax over normal crepitant pulmonary tissue is termed *pulmonary resonance*. Decreased resonance of the lower border of the lungs suggests chronic interstitial pneumonia or fibroid phthisis.

(33) A_2: represents the portion of the second heart sound caused by the sudden coaptation of the valves that guard the orifice of the aorta. This sound is best heard at the base of the heart in the second right interspace.

(34) Cardiac measurements. There is considerable variation in the size of the heart and marked discrepancies exist in the observations that have been recorded. However, the average adult heart is from 12 to 14 cm. ($3\frac{3}{4}$–6 inches) long, and from 9 to 11 cm. ($3\frac{1}{2}$ –$4\frac{1}{2}$ inches) broad. The diameter of the normal heart is usually about one-half that of the chest.

(35) Electrocardiogram: a record of the electrical changes in the heart muscle during contraction.

(36) Sinus mechanism: normal cardiac mechanism, in which the normal pace-maker or sinus node originates the heart beat rhythmically.

(37) P-R interval: the space on the electrocardiogram (from the beginning of the P-wave to the beginning of the R-wave), which indicates the length of time between the liberation of the excitation wave by the sinus node and its arrival in the ventricle (conduction time). The time is measured on curves by means of vertical lines traced at intervals of 0.04 (or 1/25) seconds. Normally the P-R interval does not exceed five small divisions, or 0.20 (or 1/5) second. P, therefore, represents the beginning of auricular activity, while Q-R-S-T are produced by activity of the ventricle. P = systole of auricles; P-R = conduction time; R-T = systole of ventricles; T-R = diastole of ventricles; T-P = diastole of entire heart.

(38) Leads I, II, and III. *Lead I.*—The record secured when the right and left arms are connected with the electrocardiograph. *Lead II.*—The

record secured when the right arm and left leg are connected with the electrocardiograph. *Lead III.*—The record secured when the left arm and left leg are connected with the electrocardiograph.

(39) Mitral insufficiency: incomplete closure of the mitral valve, occurring as a result of organic deformity of the valve segments, whereby the border of the cusps cannot be brought into close coaptation during ventricular systole. The blood is therefore permitted, by an incompetent valve, to regurgitate into the left auricle, and a systolic murmur becomes audible upon auscultation.

(40) Mitral stenosis: a narrowing of the mitral orifice, which prevents the free flow of blood from the left auricle to the left ventricle, causing a vibration or murmur, especially when the left auricle contracts just before ventricular systole (pre-systolic murmur), forces the blood through the narrowed valve.

(41) Enlargement of the liver. In the normal adult subject, the inferior border of the liver does not extend below the costal margin. Obstruction of the common bile duct is one of the causes of enlargement of the liver.

(42) Abdominal tenderness. When tenderness is elicited on palpation of the surface of the abdomen, the possibility of a diseased abdominal organ is indicated. The tenderness is most apt to be encountered over the gall bladder, stomach, spleen, kidney, appendix, and sigmoid flexure. In acute peritonitis there is general or diffused tenderness.

(43) Abdominal rigidity. Rigidity of the abdominal wall with possibly spasm, upon attempts at palpation, indicates inflammation of the peritoneum or of an abdominal organ. Rigidity of the right rectus muscle alone occur with acute appendicitis, whereas bilateral rigidity of the recti muscles accompanies acute peritonitis.

(44) Abdominal masses. During palpation of the abdomen the existence of new growths may be determined.

(45) Blood-pressure: the force exerted by the blood upon the vessel walls, which is expressed in millimeters of mercury in a standard column. Systolic pressure is the pressure of the blood during ventricular contraction (systole); diastolic pressure, during ventricular relaxation (diastole). In the normal adult, systolic blood pressure may be estimated by adding 100 to the patient's age and expressing it in millimeters. The blood pressure here recorded is low for this individual.

(46) Urinalysis. Normal urine is usually yellow or reddish-yellow in color. It is clear, and slightly acid in reaction. The normal range in specific gravity is from about 1,017 to 1,020. Albumin, sugar, acetone, diacetic acid, and indican are abnormal constituents of urine. Few epithelial cells are found in normal urine.

(47) Hematology. Blood normally contains the following cellular elements: red blood cells, 5,000,000 per cmm.; white blood cells, 7,500 per cmm.; leukocytes, differentiated mainly into (a) polymorphonuclear neutrophilic leukocytes, 60–70 per cent; (b) lymphocytes, 25–33 per cent; (c) endotheliocytes, eosinophilic leukocytes, and basophilic leukocytes occur in small numbers in normal blood. All inflammatory and suppurative processes cause a degree of leukocytosis, dependent upon the severity of the infection, and the resistance of the individual. Arneth was the first to suggest grouping of the neutrophilic leukocytes into classes according to the number of lobes in their nuclei. This is really a classification of the neutrophils according to age, the younger forms being "non-lobulated." The clinical value of this classification is not yet definitely determined, but it is believed that the first reaction to infection is an increase in the non-lobulated cells. Therefore an "Arneth count" may be a more sensitive indication of infection. Normally the non-lobulated cells constitute 35 to 40 per cent of the neutrophilic leukocytes.

(48) Blood chemistry. Blood normally contains certain constituents of diagnostic significance in problems of metabolism and excretion. The waste products urea, uric acid, and creatinin, are of chief importance from the clinical point of view. The amounts present in the blood in health are as follows (per 100 cc.): Total non-protein nitrogen, 25–30 mg.; urea nitrogen 12–15 mg.; uric acid 2–3 mg.; creatinin 1–2 mg. Higher figures are usually referable to accumulation of the respective substances in the blood because of defective elimination. In health each 100 cc. of blood contains about 100 mg. of sugar (dextrose). One of the most reliable means of detecting acidosis, and measuring its degree, is to determine the capacity of the blood plasma to combine with carbon dioxide. This test is, therefore, included as a routine determination in the chemic examination of the blood. The normal range is 53 to 75 cc. of carbon dioxide per 100 cc. of blood. Figures below 50 per cent in adults indicate acidosis; below 30 per cent, severe acidosis.

(49) Blood-complement fixation test. Wassermann test for syphilis.

(50) Kahn test: a less complicated test for syphilis than the Wassermann reaction, and believed to be as reliable.

(51) Blood culture: a procedure to ascertain the possible presence of living bacteria in the blood. The specimen for examination consists of 10–20 cc. of blood withdrawn from a vein with a sterile needle. The negative report in this case indicates that there was no general septicemia.

(52) Phenolsulphonephthalein test: the intramuscular or intravenous injection of a solution of phenolsulphonephthalein, a drug eliminated only by the kidneys, and whose amount in the urine is easily estimated by colori-

metric methods. The time of its earliest appearance in the urine, and the quantity eliminated within a definite period, are taken as measures of the functional capacity of the kidneys. Under normal conditions the drug appears in the urine in five to eleven minutes after the injection. Within the first hour after its appearance, 40 to 50 per cent is eliminated. If in two hours a total of 60 to 75 per cent is eliminated, the kidney function is considered normal. Pathologically, the excretion may be reduced to a trace, or in extreme cases to none at all in the two hours.

(53) Mosenthal test. Mosenthal placed upon a practical basis a valuable test of kidney function based upon the characteristics of the urine, chiefly the volume of the night urine and variations in specific gravity of two-hour specimens taken during the day, when the patient is upon a prescribed diet. In health the night urine, much less than the total day urine, is usually 250 to 350 cc. The highest specific gravity recorded for the two-hour day specimens exceeds 1,018. The difference between the highest and the lowest is not less than 8 or 9 points.

(54) Stage operation. In order to avoid the sudden augmentation of the general absorption of toxins, which may result from the removal of many infected teeth at one operation, it is advisable to perform the removal of infected teeth in several stages.

(55) Exacerbation: increase in the severity of a disease or its symptoms.

(56) Subjective symptoms: those that are apparent only to the patient.

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667 Madison Avenue.

DENTAL EDUCATION: A FACTOR IN PUBLIC WELFARE[1]

WILLIAM J. GIES, PH.D., F.A.C.D.

Carnegie Foundation for the Advancement of Teaching, New York City

AND

JAMES SULLIVAN, PH.D.

Assistant Commissioner for Higher and Professional Education, Department of Education, Albany, New York

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I

WILLIAM J. GIES, PH.D., F.A.C.D., CARNEGIE FOUNDATION FOR THE ADVANCEMENT OF TEACHING, NEW YORK CITY

A. INTRODUCTION

Before considering dental education as a factor in public welfare, I shall refer briefly to some common terms. Dentistry is a natural division of "*health service.*" This term, which seems to have an unmistakable meaning, obviously refers to any measure, art, science, or all of them, employed to *keep people well* or to restore their health when they are not well. Dentistry, in this view, is primarily a method to promote public welfare by advancing the physical and mental well being of the individual person. Dentistry is "*oral health-service,*" which, as a descriptive term, is synonymous with "*stomatology.*" For laymen, oral health service is not a term of mystery like stomatology.

Dentistry achieves health service largely by mechanical and esthetic means, which are being steadily improved. Dentistry must also attain this purpose with increasing efficiency in its medical agencies.

[1] Addresses at a meeting of the First Distrist Dental Society of the State of New York, New York Academy of Medicine, November 7, 1927.

Oral health-service may be said to stand on a tripod, the legs of which are medicine, mechanics, and esthetics, and the platform of which is hygiene, the legs being equal in length although not in thickness. By this figure of speech I mean that each of the three primary phases of dental practice must be sufficient in quality to keep the whole system of oral health-service balanced and adequate for the patient, but the proportion of each phase need not be the same either in practice, or in education for practice. And of course normal conditions, and hygienic measures to maintain or restore the normal—prevention, in short—are the alpha and omega of every division of health service.

I use "health service" as a synonym of the term "medicine" in the academic sense—the broadest modern sense—of "medicine." Originally "medicine" meant remedy; now the most significant part of its import is quite the contrary—*to make remedial or curative treatment unnecessary.* I distinguish between "medicine" and "*practice of medicine,*" the former being a general concept of the whole of health service; the latter, a special concept of only a part. In the most general sense, "medicine" is health service in all its divisions, among which is the part that may be practised in, say, New York by a licensed person who has received the degree of M.D. from a registered medical school. But "medicine" in its most general sense also includes the practices of dentistry, nursing, midwifery, community health education, public health administration, pharmacy, optometry, chiropody, etc. All of those who practise any of the arts implied by these terms are practitioners of "medicine" in the sense that they are practitioners of *health service.* But only those who receive the M.D. degree may practice "medicine" in the legally restricted sense of the term (which does not include dentistry), and these alone are "physicians." While the statutes remain as they are, dentists are not and cannot become physicians unless they obtain the M.D. degree and pass license examinations for admission to the legalized practice of medicine. And physicians are not and cannot become dentists unless they obtain the D.D.S. or D.M.D. degree and pass license examinations for admission to the legalized practice of dentistry. Much confusion in thought, discussion, purpose, and action arises from indifference to these obvious distinctions.

If it be conceded that dentistry is a division of health service—a part of medicine in the broadest sense of the term—then the greatest usefulness of dental education in any phase must arise from the promotion of oral *health* in the individual person through self-help, or of oral *health service* by the practitioner for the patient, or both. Considered from this general standpoint, four different types of dental education may be noted: (a) dental education of the public; (b) dental education of the prospective dentist; (c) dental education of practising dentists; and (d) dental education of physicians. Successful dental education of each of these four groups would greatly promote the further development of dentistry as a factor in public welfare.

B. FOUR TYPES OF DENTAL EDUCATION SELECTED FOR GENERAL DISCUSSION

(*a*) *Dental education of the public*

Dental education of the public, to be most effective, should be included in or associated with procedures for general health education, which ought to inform the individual in simple and understandable terms regarding the habits he should develop in order to preserve or improve his health and the health of all parts of the body. Such health education should also help him to act wisely for the restoration of health when he is ill. This type of popular health education, including dental education, can be promoted very effectively not only by physicians and nurses, but also by dentists and dental hygienists, in the offices of practitioners, in public schools, at meetings of parent associations, and through other means of professionally responsible publicity; also by dental societies, by dental schools in their infirmaries, by health departments, etc. It is essential, however, if the dental profession would not be misunderstood and degraded, that popular dental education should be sincere, authoritative, non-technical, and conducted unmistakably for the benefit primarily of the public. Dentistry is a profession, not a trade. Intelligent laymen readily surmise the import of plans of publicity intended chiefly to increase business for commercial practitioners, and draw damaging conclusions that soon become widespread.

Dental education of the public, under any of these auspices or by

any of these agencies, should include not only the conventional guidance relating to direct care of the teeth, such as oral hygiene, but also hygienic instruction on the less obvious influences, such as dietary deficiencies, customs and habits, occupations, and prenatal conditions, on teeth and their development. Non-technical knowledge of this kind would enable parents to improve their own general and oral health and, so far as these conditions are influential and controllable, would help pregnant mothers to produce healthy children. It would encourage parents to teach their children habits and measures that would promote general health and the preservation of the teeth.

In dental education of the public, dentists for many years have performed a very influential service for the prevention not only of dental disease but also of disease in general by encouraging patients to consult recurrently for the detection and treatment of dental disorders in their incipiency. Dentistry has been far in advance of medicine in this respect. Medicine has made great contributions to community welfare by providing protection against such diseases as small pox, yellow fever, and other plagues. But in *private practice* physicians have not been equally alert in promoting the welfare of the individual by preventive service to *keep* him well. Dentists, by encouraging patients to visit them frequently for the early discovery of deficiencies, have set an example in periodic health examination that should spread to private practice in all divisions of health service.

In the dental education of the public, it is important to promote understanding of the fact that philanthropy is essential for the advancement of dentistry and the education of dentists. It is remarkable that the income from endowment in our dental schools does not average much more than $1000 per school per year. The gifts for dental education, compared with the gifts to "medicine," have been few and far between. The public has shown very little interest in the promotion of dental research and dental education, apparently because it has not realized the intrinsic importance of dentistry as health service, or assumes that "medicine" is promoting oral health-service. Of recent gifts, the largest was that by Mrs. Montgomery Ward of Chicago. As a result of her generosity the Northwestern University Medical and Dental Schools are associated in a new and exceptional building under conditions that favor the accomplishment of their

important work Gifts such as those by the Forsyth Brothers and Mr. Eastman, in support chiefly of clinical service, are also among the most useful for dental education of the public. Individual dentists, dental societies, and dental schools should show, with increasing persuasiveness, the reasons why dentistry cannot continue to develop adequately, in the public interest, without large gifts of funds for the purpose. When the public receives the right kind of dental education it may be expected to do for dentistry relatively what it has for medicine, and to promote the public welfare effectively through dental agencies.

(*b*) *Dental education of the prospective dentist*

Dentistry has suffered somewhat in the past, when compared with other professions, because it has been based on a relatively low plane of general education. Individual dentists, without going to an academic college or to a dental school, have attained distinction as practitioners. Careful observation, close application, reflection, personal ambition, industry and devotion to ideals, in the gifted man, enable him to teach himself. But these qualities are no longer sufficient, and graduation from a dental school is essential for admission to practice. If we regard the academic college as affording *opportunities for self development*, then most men who obtain this more advanced education will go farther in the dental school and in practice than without such a preparatory training. When dentists are placed educationally on a parity with physicians, by the requirement of at least two years of approved work in an academic college for admission to a dental school, certain disabilities from which dentistry has suffered will be eliminated. Under such conditions there will be a transformation not only in the relations of dentistry to the other professions but also within dentistry itself, in increased personal self-respect; in the consciousness of greater professional insight, ability, and usefulness; and in the development of a larger number of influential teachers, investigators, and leaders.

There is another important aspect of training for the practice of dentistry to which little attention is given. The dental school should concentrate, in its general curriculum, on the training of the general practitioner. It should not attempt *in the same courses*, as is now the custom, to train the prospective dentist to be a general practitioner

and also every kind of specialist. It has become impossible to teach all that might be told a student. The young dentist cannot be as competent a practitioner, at graduation, as he may be at fifty. But, despite these facts, the tendency of most teachers is to try to tell the student "everything" in order to make him as expert at graduation as each of his teachers. Let him be trained in the undergraduate years to be first of all a capable, reliable, *general* practitioner so that he will be able to begin a dependable practice; and be so imbued with ardent desire to continue to be a student—so filled with the dignity, opportunity, and responsibility of his profession—that through self-instruction and experience he will grow steadily in capacity and proficiency. The attempt to achieve more than this in the undergraduate curriculum accounts for some of the deficiencies in the present system of education in the dental schools.[2]

[2] In the Carnegie Foundation's Bulletin on Dental Education the writer suggested the two-three-graduate plan of dental education as particularly suitable for the training of general practitioners and of specialists, the three years of the undergraduate curriculum to be lengthened to the clock-hour content of four conventional years where that is regarded as essential. A few days before the meeting at which these addresses were delivered, the Department of Education of the State of New York announced that dental schools registered in this state may conduct the specified minimum curricular requirements in three years of four quarters each. (*Journal of Dental Research*, 1927, vii, p. 455.)

In a discussion in this relation, of proposed improvements in dental education, the author wrote in part as follows in the Bulletin on Dental Education in the United States and Canada (1926: pp. 239–240):

"This extension and improvement [to make dentistry, in the quality and efficiency of its service to the patient, the full equivalent of an oral specialty of the practice of medicine] could be accomplished without requiring the prospective general practitioner of dentistry to become a doctor of medicine before beginning his dental training, and could best be brought about by pursuit of the following three main objectives: (a) *the preliminary education and the instruction in the medical sciences should be practically the same in general scope and quality as for medicine;* (b) the technical and clinical training, the applications of the medical sciences, and the correlations of clinical dentistry with clinical medicine should be sufficient to assure both ability to initiate safely a dependable modern general practice of dentistry and capacity to grow in proficiency; and (c) the most advanced phases of dental practice should be reserved for systematic graduate study.

"These three objectives could be attained through the requirement of at least (a) two years of approved preparatory work in an accredited academic college, including several extra courses that would stimulate interest and develop ability in the prospective practice of dentistry, or reveal ineptitude, (b) and three years of intensive and well-integrated effort in an undergraduate dental curriculum for the training of general practitioners only, *the years to be lengthened by beginning them with summer sessions, or other-*

(c) *Dental education of practising dentists*

Every professional man ought to be an assiduous student. No practitioner of health service can give to patients his best attention who does not, by faithful study of the results of observation and research by others, keep fully informed regarding the developments in his field. Few men can be effective students without useful guidance. Dentists should obviously attend the meetings of their societies, to which men of ability and understanding bring new and desirable information and methods. But they should also occasionally take short, intensive, and effective courses of special instruction. These are usually given most successfully by dental societies, or in universities and in dental and medical schools.

Many men "find themselves" early in their careers and want to specialize. Dental specialists in the past, like Abraham Lincoln, the master of English, have been self-instructed and self made. They have been the aspiring men who have taught themselves by study and experience to become leaders. Give such men greater educational opportunity in the future, and specialists will become steadily more expert and useful. The university dental schools, besides providing short advanced courses for practitioners, should also offer full-year graduate curricula for the training of specialists, such as orthodontists, periodontists, maxillofacial surgeons, public health administrators, teachers, and investigators. These graduate curricula might be extended through one, two, or three years as the objectives would require. Thus, men aiming to devote their lives to dental teaching and research might wish to do graduate work for three years and receive the Ph.D. degree under the usual conditions.

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wise, wherever the time equivalent of four professional years of conventional length is regarded as essential; followed by (c) optional supplementary full-year graduate curricula for advanced training, during one or more years, in all types of dental and oral specialization. *The suggested lengthening of the dental years, which might be accomplished by their subdivision into "quarters" in the conventional manner, would prevent long interruptions in the digital training besides adding a year to the practitioner's career in practice.* Loan funds could be used to aid students in need of financial assistance." [Italic not in the original.]

(*d*) *Dental education of physicians*

It is a pleasure to have an opportunity in this building [New York Academy of Medicine], where you are guests, to note that you are welcome here and hold your meetings as you would in a home of your own. You are plainly recognized by the Academy of Medicine as fellow practitioners of health service.

Unfortunately a remarkably large number of doctors of medicine are woefully ignorant of the import of clinical dentistry and are seriously in need of education in the elements of oral hygiene and of oral medicine. This deplorable situation seems to be due largely to the fact that medical schools traditionally ignore these subjects,[8] and physicians are often too intolerant to learn from association, consultation, or experience. These regrettable conditions would probably disappear, if medical schools gave adequate attention to oral hygiene and clinical dentistry, and if both dental and medical societies, in occasional joint meetings, devoted more constructive attention to problems on the border line between medicine and dentistry. Combined curricula for the M.D. and D.D.S. degrees, for the few who might be inclined to undertake practice that included the responsibilities of both the physician and the dentist, would do much, by encouraging full appreciation of the related problems, to promote

[8] In the Carnegie Foundation's Bulletin on Dental Education in the United States and Canada (1926), the writer, alluding to this situation, wrote in part as follows (pp. 246–47):

"The unconcern for clinical dentistry at medical schools is mentioned frequently in the summaries [in Part VI of the Bulletin]. Although formal courses relating to the specialties of the eye, ear, nose, and throat are included in the undergraduate medical curriculum, analogous instruction on the maladies of the teeth and mouth are usually lacking. In emphasizing the neglect of oral health-service in medical schools it is not the writer's purpose to suggest that the present overload of specialties in the undergraduate medical curriculum should be increased. On the contrary, the integration, in the undergraduate medical curriculum, of suitable instruction in clinical dentistry with that relating to the parts adjacent to the mouth, and the reintegration of the useful content of the current courses in the specialties with those in more general relationships, would seem to be preferable. Orientation courses on the chief correlations between medicine and dentistry, such as those at the medical schools of the Universities of Minnesota and Toronto, although requiring but a few hours, seem not only to help the physician to note the occurrence and import of pathological conditions in the mouth and to comprehend the scope of dental practice, but also to prepare him for responsible action regarding dental ailments, without encouraging him to believe that he has mastered any of the specialties of oral health-service."

understanding and coöperation between practitioners in both professions for the benefit of the patient.[4]

It is highly important that dentists should endeavor more earnestly to show plainly to physicians the true relation of dentistry to human welfare. Dentists should seek to understand the conditions in medical practice that underlie oral health service, and physicians should exhibit a finer spirit of helpfulness. Antagonism between medicine and dentistry cannot be explained on any basis of public interest or advantage and has no justification in any sentiments that are worthy of respect, for both professions are agencies for health service and

[4] "The prevailing attitude of the medical faculties toward dental education may be illustrated by two typical incidents of recent occurrence. During a discussion of professional experiences, a group of medical professors in universities holding membership in the Association of American Universities enquired of one of their number, who had lately gone to a new post, how his work was progressing. 'Very satisfactorily in all respects but one,' he said: a member of his staff was 'so hopelessly inefficient that the only thing to do about it for the rest of the year was to put him in charge of the dental students.' This solution was regarded, by most of those to whom it was stated, as a suitable way out of the predicament. The victims were students at a Class A dental school. On another occasion, the dean of a school of medicine with which a Class A school of dentistry was intimately associated, and where complete rebuilding of the medical school was contemplated, when asked, 'are you planning to include the dental school in the medical centre on the new site,' replied: 'No, we are not planning to *include* the dental school, we are planning to *exclude* it.' The intended elimination has been carried to complete success, with results that may involve the extinction of the dental school. [The dental school has been discontinued.] Medical schools collectively, and schools of hygiene and public health, pay little or no attention to oral hygiene; ignore clinical dentistry; overlook the important relations of disease and abnormality of the teeth to the general health; do not even provide for training in oral surgery; teach their students to inspect a patient's tongue and the oral tissues, but to disregard his teeth; and in general, so far as dental disorders are concerned, violate not only the dictates of medical common sense but also reject such wisdom as that expressed by Sir William Osler when he wrote: 'There is not any single thing more important to the public in the whole range of hygiene than the hygiene of the mouth. If I were asked to say whether more physical deterioration was produced by alcohol or by defective teeth, I should unhesitatingly say defective teeth.' The failure to encourage and to advance the development of clinical dentistry is a regrettable phase of medical history. When it is recalled that a generous and confiding public, following the promptings of medical leadership, has given to medicine enormous sums of money intended to support impartially and without prejudice all suitable efforts to keep people well, the indifference in medical schools to the diseases of the teeth and mouth and their systemic sequelae, and to the promotion of medical understanding and competency in those who aim to devote themselves professionally to oral health-service, cannot be regarded as reasonable or worthy." (Gies: Carnegie Foundation's Bulletin on Dental Education in the United States and Canada, 1926, p. 136.)

cannot render it faithfully on any other conditions than those of earnest and effective coöperation.

I hope that, in the development of each of the four general types of dental education to which I have briefly alluded, barriers that embarrass understanding and coöperation among practitioners will be removed; that research and teaching, promoted by generous public support, will rapidly solve one serious health problem after another; and that all concerned will derive abiding contentment from health service in which prevention of disease, of distress, and of disability will become cumulatively more successful and widespread.

II

JAMES SULLIVAN, PH.D., ASSISTANT COMMISSIONER FOR HIGHER AND PROFESSIONAL EDUCATION, DEPARTMENT OF EDUCATION, ALBANY, N. Y.

I was very much pleased to be asked to speak to you for the main reason that I wished to meet you. I have recently taken the place of Dr. Downing as the Commissioner for Higher and Professional Education. Some of my friends say I am more comparable to Mr. Gompers or to Mr. Green, in charge of a sublimated form of trades union organized by the State and effectively enforcing the law for the benefit of the profession. But I was interested in meeting your members because there is value in personal contact. I immediately found that some of them were friends of mine—that one was a great friend of Mr. MacAndrew in Chicago, who is trying to set Mayor Thompson right.

I feel that Dr. Gies has so effectively covered the field that has been laid before me that I am going to touch on only some phases of it. My memory for dental education goes back to a time when it was not respected. I remember that, when I was looking forward to going to college to take the degree of doctor of philosophy, my fellow-students in a high school in Boston were not then required to complete three years of high school for admission to a dental college. Just one year later they *were* called upon to complete three years of high school, and could enter a dental college and become dentists with the title of "Doctor" at the end of three years—and there was only a six-months course in each year. Whereas, to get the Ph.D. degree,

I was called upon to spend four years in high school, four years in college, and then three or possibly four years of graduate study.

It was inevitable, that, under such conditions, the dental profession did not enjoy very high repute educationally. From that time, which was about the beginning of the early nineties, to the present, as dental education has required more and more preparation, it has won more and more respect from the public. If you are going to make any profession reputable, you must make the educational attainments high. If you would make them equal one to another, then the educational requirements must be on the same level or the public will not give them the same respect. If you are going to make dentistry coequal with medicine in public respect, then your dental requirements must be equal to those of medicine. I don't think that is a subject of dispute.

I do not know the details of the requirements of the dental profession. I am a mere amateur compared with a man like Dr. Gies. But I have been in dental schools and medical schools enough to realize that one of the serious defects to be overcome is what I should call the insufficient preparation of the highly professional man in the matter of presenting his subject. Our institutions on this side of the Atlantic are organized in a somewhat different way from those on the other side. Over there there is more of an inclination to use competitive methods of instruction. For example, at the University of Berlin one learns about the "Privatdocent," who is a man in competition with his colleagues to get students on the merit of the work he is giving. In our country we are altogether too prone, both in the academic and professional work, to appoint men to teaching positions because of their high academic attainments, or their high professional attainments. Sometimes we pay altogether too little attention to the quality of these men as instructors. A man may be an excellent painter, but he may know very little about imparting his knowledge to students. And I think that has been more true of our professional schools—law, medicine, dentistry—and also of our academic collegiate work, than it has been of the elementary institutions. So if you were to ask me what defect I have found, and I do not confine my remarks to dentistry, I would say there is a well-marked defect in the matter of efficient instruction.

Naturally the public takes an interest in instruction in a somewhat indirect fashion. The more you require of a professional man in the way of preparation for his job, naturally the more he must exact from the public as a return for his preparation. If it is going to cost him $10,000, and the lack of money-earning power for the years that he is studying, then his demand on the public purse is going to be large. Therefore, in our effort to accomplish certain kinds of work in dentistry, or to accomplish certain kinds of work in what we call medicine, the question arises: Is the same preparation necessary for all people who go into that particular portion of health work?

A short time ago, in this city, there was a very alluring proposition of a single-salary schedule for teachers. The kindergarten teacher was to receive as much—the superintendents themselves excluded—as any other teacher below a high-school principal. In other words, they were going to require a Ph.D. education of a teacher who only needed what we called a kindergarten teacher's salary. If that were done, then you would have to pay Ph.D. salaries to kindergarten teachers. If you are going to compel an optometrist to have a medical degree, then that optometrist must be entitled to charge a physician's prices. There has come into the dental profession a very laudable development of the dental hygienist, who can do a certain kind of work in connection with dentistry, but is not qualified to do the work of the well prepared dental surgeon. The only trouble sometimes is that when a dental hygienist is employed to do this kind of work, the bill goes out to the public as if the dentist had done the work, which, of course, is not a very acceptable form of bill to the public. At the same time, I do not think we can do anything except regard dental education as a phase of medical education, just as I would say that a man who is a specialist in diseases of the eye, ear, throat, and nose, or the oculist, or the aurist, is practising a specified branch of medicine. I feel that you men are physicians with a specialty.

I am going to turn from that phase of your dental work—from the education of the dentist, which Dr. Gies has handled very much more completely than I could hope to—to the dental education of the public. That he did not handle in quite the same way that I should, for the reason that I have had many years of experience with children and their education.

I include in my thought children all the way from college grade right down to the first years of elementary school. There is the place for preventive dentistry. It is a phase of education in which we wish to bring home to the public the necessary precautions that should be taken with the teeth of their children. I confess it is a hard job. In the first place, we are fighting human temptations all the time. We preach against carelessness about diet, avoidance of excessive eating of sweets, etc. It almost discourages one to feel that most of it goes into one ear and out the other. The human creature is always willing to "take a chance," especially on his life and his health. You will find that practically one hundred per cent insure their houses, but not more than fifty per cent insure their bodies. That is the phase of the human equation that we are constantly fighting, and which is so discouraging in health work or public welfare-work with children.

Of course there has been great enthusiasm in this field. It reached its apex about the year 1917, after Dr. Fones had performed some very interesting experiments in Bridgeport with his group of dental hygienists. We felt the effect of that experiment in this city. We began a campaign for public health-work. But we did not get the kind of health work in the schools that we wanted. In your city this work is under the control of the Board of Health, not under the control of the schools. At that time, I was just beginning the principalship of the Boys High School in Brooklyn, and was particularly enthusiastic about the care of the teeth. It had been a disagreeable experience to see children with their teeth in poor condition, so the Board of Health sent me a physician. The physician reported daily, signed his name in a book, and ran away as rapidly as possible.

I did not regard that procedure as proper health inspection, but it was so designated by the Department of Health. This physician said: "I expect your teachers to pick out the children and send them down to me." I said: "Do you think that any layman, busy with his particular subject, is able to diagnose dental conditions in children he sees for only forty-five minutes each day?" I had an acrimonious discussion with the Health Commissioner, not with the City Superintendent who sympathized with me. To the Board of Education I said: "If I am going to get that kind of health inspection, I don't want it at all." They inquired: "What do you want?" I replied: "I want someone who has a knowledge of medicine, or a knowledge of

oral hygiene." So they sent me a nurse, and I put her in the gymnasium. It was a school exclusively for boys. I set her to work examining the mouths of all the boys. She polished them up, not only orally but generally. She got them to take care of their teeth, and to go to dentists for more serious conditions. They disliked, at a second examination, to be reprimanded for having done nothing. She cleaned them up as to their clothing. A boy associating with boys is not likely to be so careful about his appearance as when he has to pass the inspection of an attractive nurse. (I saw to it that she was *attractive*).

The problem is not only with the high school boy, but also with the elementary school boy, and with the collegian. I have known graduate physicians who took "a chance" on the contraction of disease, even when they knew the results of it—because they are human beings.

I am going to talk just a few minutes more on the subject of public welfare. I take up this subject with considerable diffidence because it touches your pocketbooks. You know there is nothing that so excites a man, whether he is a college professor, a physician, a dentist, or a member of any other profession, as diminution of his income. But it is a phase of the public welfare which is becoming extremely oppressive. When you hear men about a club, a country club or the golf links, say that such and such a person or group of persons in a profession are a "bunch of highway robbers" because of excessive charges, something is wrong. You know there is a saying in economics that when taxation exceeds the effective desire of accumulation, people won't save money. We have to a certain extent made a fetish of some kinds of medicine. An operation for appendicitis does not perform itself. It must be done by a surgeon. But in certain parts of the country, I will not say the City of New York, our medical profession has led the people to believe that child bearing is a pathological instead of a physiological process. Millions of women have borne children but never had any care by a physician. We have put out the midwife; she was ignorant, untutored. But we have given the public no one to take her place except a very high priced physician. I maintain that it is contrary to public welfare if the charge for professional service has reached such a point that it deters the middle classes from having children—and it *has* reached that point. It is a dangerous state of civilization where women, anxious to have

children, feel that they cannot because of the excessive expense connected with child bearing in a great city like New York. I don't remove any responsibility from these women. I realize that they will spend any amount of money on theaters; that the general motto among the poorer groups is: "The State will take care of my necessities; I will take care of the luxuries."

On Third Avenue I have watched women—slattern, their shoes worn out, barely a mackintosh to cover their bodies—giving up their dole at the vaudeville window, and then going to the dispensary for free medical treatment. These people are now being better taken care of, medically, than the middle classes. The wealthy take good care of themselves. The people who are being injured by excessive charges are those of the middle classes. Where can they go? They can't plead poverty—they have too much self-respect for that. They deprive themselves of what they cannot afford. I recommended, to an orthodontist, a friend of mine whose daughter, at the age of six, had lower incisors that were coming out slighty in front of her uppers. He took this child to the orthodontist and asked what the treatment would cost, and was told $1200. That was said to a man who was receiving $1800 a year as a teacher. Do you realize the effect of that kind of thing on such a man? He would have to neglect his child's teeth because he could not pay the excessive charge.

I told this to one of your profession in Brooklyn, and he said: "I don't do that kind of work ordinarily, but have him send his daughter to me and I will look after her." He did so, and the bill was only $75. I know the young lady. She has a very beautiful set of teeth, and it did not cost her poor father $1200 to assure it. Get the effect of excessive charges on the public welfare through the reaction of the individual. He is going to say: "I can't afford it. It would put me in the poor house. When artificial teeth are needed, I will get them all at one time, and that will be the end of it."

I mention these things, although with a good deal of diffidence, because they are related to the solution of a very important problem in the promotion of the public welfare. It is one that dentists will have to consider. I am not making a plea for your wealthy patients, or for the poor. I am asking your consideration for the great class that lies between them. They ought to be the class to provide the population in this country, but at present they are not.

BIOCHEMICAL STUDIES OF STREPTOCOCCI. II. AVAILABILITY OF 671 SYNTHETIC MEDIA[1]

FRANCES KRASNOW, PH.D., AND MARGARET L. ROSENBERG, M.A.

Laboratory of Biological Chemistry of Columbia University, at the College of Physicians and Surgeons, New York

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I. INTRODUCTION

Synthetic media have been employed in various researches on the nutrition and metabolism of microörganisms. In a previous publication, these investigations have been summarized on the basis of organisms studied.[2] The list was not complete, but it indicated the typical researches in the field.

II. COMPOSITION OF MEDIA AND DATA ON THEIR AVAILABILITY

A. Types of media. Instead of devising new combinations of nutrients, we used, in the greater number of the 671 media tested, those described by previous workers. This plan was followed because we hoped to correlate more readily our findings with theirs. It was impossible to duplicate a number of media because very few recipes are described in any detail, and in some cases the names of the chemicals used were not recorded.

In the present work (a) all ingredients are given in detail. (b)

[1] The first paper in this series was published in the last preceding issue of the *Journal of Dental Research*, 1929, ix, p. 29.

[2] Krasnow, Rivkin, and Rosenberg. *Proceedings of the Society for Experimental Biology and Medicine*, 1925, xxiii, 215; also *Journal of Bacteriology*, 1926, xii, 385.

Wherever reactions were adjusted with $NaHCO_3$ or K_2CO_3, the amounts of these substances were accurately determined and recorded in the tables. (c) Aspartic acid was substituted for asparagin or sodium asparaginate (20). (d) The term "trace," so often used to designate small quantities, is assigned a definite value; namely, 0.1 cc. of a 0.1 per cent solution (or 0.1 per cent suspension in cases of slightly soluble substances) per 100 cc. of medium—0.0001 per cent. (e) The numbers of the media given in the first columns of the tables are our serial numbers.

B. Media and results. The 671 media with their corresponding results are listed in a series of thirty-four tables. In general, each consists of media suggested by one investigator, or by a group of investigators using related media. *Tables 33* and *34* give the data for miscellaneous media.

C. Reading of results. The results recorded in the tables consist only of those necessary to show the comparative values of the various media. The symbols, 4+, 3+, 2+, +, several colonies, c, and 0, are used to indicate the amount of growth; 4+ denotes the maximum. Therefore the records for the subcultures of the last four transplants from all protocols except one were omitted. However, the tests on each medium included six transplants. The one from the test suspension to the synthetic medium is designated as the first transplant; the five others are successive transplants from synthetic medium to synthetic medium. The subcultures from each transplant numbered one more than the one immediately following; thus, there were six subcultures from the first transplant, five from the second, four from the third, three from the fourth, two from the fifth, and one from the sixth.[3] See *Tables 1–35*.

III. DISCUSSION OF RESULTS

A brief survey of the data recorded in this investigation of 671 media shows that 441 permitted the streptococci to remain viable through one or more transplants. While this would seem to indicate that it is a comparatively simple matter to prepare synthetic mixtures for the maintenance of these organisms, our results show that all of

[3] Details of procedure appear in the *Journal of Bacteriology*, 1926, xii, 385.

these media were markedly deficient. The most significant data are summarized in *Table 35*.

These data and *fig. 1* indicate (a) decrease in the proportion of media showing live organisms in successive transplants; (b) very

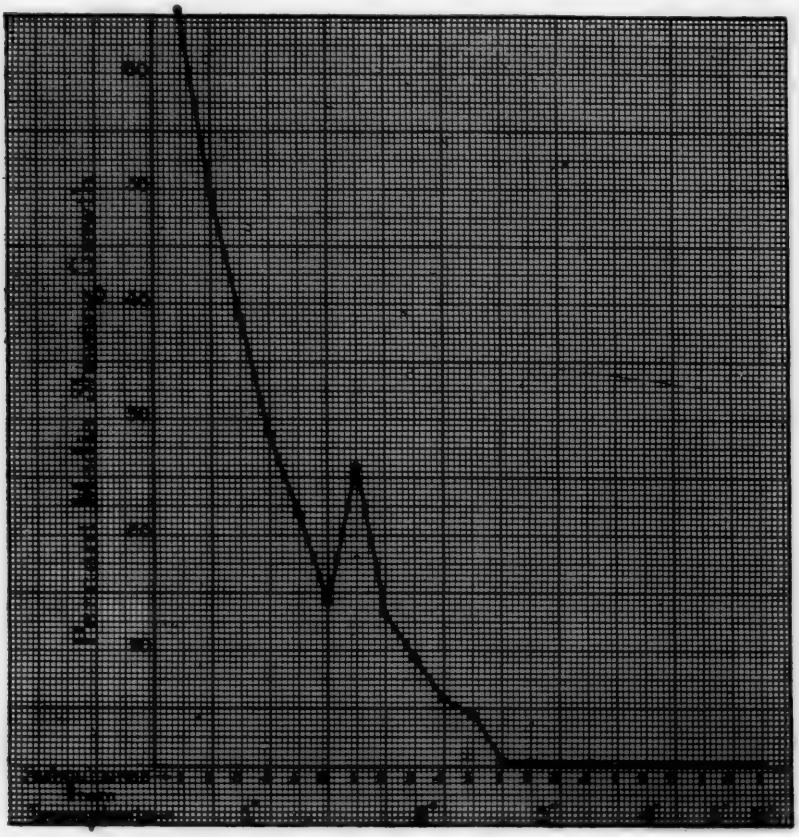

FIG. 1. Graph showing decrease in the nutritive quality of synthetic media for successive transplants of streptococci.

marked falling off in the number of "positives" in the successive subcultures of the first transplant series; (c) change that is not so abrupt in the second transplant series, (d) only one medium supporting

growth in the third transplant series, and (e) no medium showing growth in the fourth, fifth, or sixth transplants.

The media do not permit the physiological functions of this organism to continue normally, hence propagation decreases and finally ceases. Continued growth in the first two transplants may be due either to the small reserve store of the necessary nutrient (nutrients ?), or to the transfer of small amounts of some of the constituents of the conventional media on which the test culture was amassed—this, despite our extreme care to obtain a pure suspension. The amount of substance so transferred could not have been more than that adhering to the bacterial bodies.

IV. TABLES

TABLE 1

Aubel media

NUMBER OF MEDIUM	BIBLIOGRAPHY	NaOH 2.123M		PERCENTAGE COMPOSITION						RESULTS OF GROWTH										
										First transplant						Second transplant				
										Subcultures:						Subcultures:				
		Cc. per 5 cc. before sterilization	Cc. per 1 cc. after sterilization	NH_4Cl	Aspartic acid	$CaCO_3$	$MgSO_4 \cdot 7H_2O$	$MnSO_4 \cdot 4H_2O$	KH_2PO_4	1	2	3	4	5	6	1	2	3	4	5
49	3	0.023		0.5		0.1	0.1		0.1	0	0	0	0	0	0	0	0	0	0	0
49*a*[1]	3	0.03	0.004	0.5			0.1		0.1	0	0	0	0	0	0	0	0	0	0	0
74	2	0.1			0.5		0.1[2]		0.1	3+	4+	4+	4+	0	+	+	+	+	0	+
431	2	0.07	0.01		0.5			0.1	0.1	3+	2+	0	0	0	0	0	0	0	0	0

[1] Adaptation of 49—$CaCO_3$ omitted.

[2] Adaptation of 431—$MgSO_4$ instead of $MnSO_4$.

TABLE 2

Ayers-Rupp media

NUMBER OF MEDIUM	BIBLIOGRAPHY	NaOH 2.123M	PERCENTAGE COMPOSITION						RESULTS OF GROWTH					
									First transplant					
									Subcultures:					
		Cc. per 5 cc. before sterilization	$NH_4NaHPO_4 \cdot 4H_2O$	Aspartic acid	Dextrose	Lactose	Na acetate.$3H_2O$	KH_2PO_4	1	2	3	4	5	6
6	4	0.055	0.4			1.0		0.2	2+	0	0	0	0	0
87	5	0.4	0.36	0.12				0.12	0	0	0	0	0	0
88	5	0.02	0.36		0.5			0.12	2+	0	0	0	0	0
156	5	0.019	0.36				0.12	0.12	0	0	0	0	0	0
157	5	0.17	0.36		0.5		0.12	0.12	0	0	0	0	0	0

TABLE 3

Beijerinck media

NUMBER OF MEDIUM	BIBLIOGRAPHY	NaOH 2.123M	PERCENTAGE COMPOSITION	RESULTS OF GROWTH										
				First transplant						Second transplant				
				Subcultures:						Subcultures:				
		Cc. per 5 cc. before sterilization		1	2	3	4	5	6	1	2	3	4	5
166	11		NH_4Cl, 0.01; $MgCl_2$, 0.01; K_2HPO_4, 0.02; $NaHCO_3$, 0.1; $Na_2S_2O_3 \cdot 5H_2O$, 0.5	0	0	0	0	0	0 0	0	0 0	0	0	0
167	11		$CaCO_3$, 2.0; KNO_3, 0.01; K_2HPO_4, 0.02; Na_2CO_3, 0.02; sulphur, 10.0	4+	4+	4+	3+	3c	3+	+	5c	0	0	0
168	11		$CaCO_3$, 2.0; $MgCl_2$, 0.01; KNO_3, 0.05; K_2HPO_4, 0.02; Na_2CO_3, 0.02; sulphur, 10.0	4+	4+	3+	+	+	+	+	3c	0	0	0
169	11		K_2HPO_4, 0.01; $NaHCO_3$, 0.02; $Na_2S_2O_3 \cdot 5H_2O$, 0.5	0	0	0	0	0	0	0	0	0	0	0
433	9	0.01	$NH_4NaHPO_4 \cdot 4H_2O$, 0.2; KCl, 0.05	+	0	1c	0	0	0	0	0	0	0	0
434[1]	10	0.05	Aspartic acid, 0.5; dextrose, 3.0; $MgSO_4 \cdot 7H_2O$, 0.01; KH_2PO_4, 0.01	+	5c	0	0	0	0	0	0	0	0	0
435	8		Aspartic acid, 0.5; $FeSO_4$ $(NH_4)_2$ $SO_4 \cdot 6H_2O$, 0.1; K_2PO_4, 0.25; Na_2CO_3, 0.25; NaCl, 0.25; Na_2 oxalate, 1.0	2+	0	0	0	0	0	1c	0	0	0	0
436	7	0.042	Aspartic acid, 0.025; K lactate, 0.1; K_2HPO_4, 0.05; $FeSO_4(NH_4)_2SO_4 \cdot 6H_2O$, trace	2c	0	0	0	0	0	0	0	0	0	0

[1] Cc. of NaOH (2.123M) per 1 cc. after sterilization = 0.006.

TABLE 4

Braun and Cahn-Bronner media

NUMBER OF MEDIUM	BIBLIOGRAPHY	NaOH 2.123M: Cc. per 5 cc. before sterilization	PERCENTAGE COMPOSITION	RESULTS OF GROWTH: First transplant: Subcultures: 1	2	3	4	5	6
61	16	0.12	NH_4 lactate, 0.6; K_2HPO_4, 0.2; NaCl, 0.5; $NaHCO_3$, 2.0	0	0	0	0	0	0
600	16		NH_4 lactate, 0.6; K_2HPO_4, 0.2; NaCl, 0.5; Na_2SO_4, 0.2; $NaHCO_3$, 2.012	0	0	0	0	0	0
601	16		NH_4 lactate, 0.6; $CaCl_2$, 0.05; K_2HPO_4, 0.2; NaCl, 0.5; $NaHCO_3$, 2.544	0	0	0	0	0	0
602	16		NH_4 lactate, 0.6; $MgSO_4 \cdot 7H_2O$, 0.1; K_2HPO_4,0.2; NaCl, 0.5; $NaHCO_3$, 2.06	0	0	0	0	0	0
603	16		NH_4 lactate, 0.6; $FeCl_3 \cdot 6H_2O$, trace; K_2HPO_4, 0.2; NaCl, 0.5; $NaHCO_3$, 1.4638	0	0	0	0	0	0
604	16		NH_4 lactate, 0.6; $Fe_2(SO_4)_3$, trace; K_2HPO_4, 0.2; NaCl, 0.5; $NaHCO_3$, 2.391	0	0	0	0	0	0
605	16		NH_4 lactate, 0.6; $CaCl_2$, 0.05; $MgSO_4 \cdot 7H_2O$, 0.1; K_2HPO_4, 0.2; NaCl, 0.5; $NaHCO_3$, 2.3858	0	0	0	0	0	0
606	16		NH_4 lactate, 0.6; $CaCl_2$, 0.05; $FeCl_3 \cdot 6H_2O$, trace; $Fe_2(SO_4)_3$, trace; $MgSO_4 \cdot 7H_2O$, 0.1; K_2HPO_4, 0.2; Na_2SO_4, 0.2; $NaHCO_3$, 2.0856	0	0	0	0	0	0
609	16	0.02	$(NH_4)_2SO_4$, 0.6; dextrose, 0.5; K_2HPO_4, 0.2; NaCl, 0.5	0	0	0	0	0	0
612	16	0.01	$(NH_4)_2SO_4$, 0.6; sucrose, 0.5; K_2HPO_4, 0.2; NaCl, 0.5	0	0	0	0	0	0
613	16		K_2HPO_4, 0.4; NaCl, 0.5; Na lactate, 0.6; $NaNO_3$, 0.5; $NaHCO_3$, 0.0996	0	0	0	0	0	0
614	16		Alanine, 0.5; K_2HPO_4, 0.2; NaCl, 0.5	0	0	0	0	0	0
615	16		Glycocoll, 0.5; K_2HPO_4, 0.2; NaCl, 0.5	3+	0	+	0	0	0
617[1]	16	0.06	Aspartic acid, 0.5; K_2HPO_4, 0.2; NaCl, 0.5	3+	0	0	0	0	0
618[2]	16	0.07	Glutamic acid, 0.5; K_2HPO_4, 0.2; NaCl, 0.5	+	0	0	0	0	0
619	16		Tryptophane, 0.5; K_2HPO_4, 0.2; NaCl, 0.5	0	0	0	0	0	0
599	16	0.053	KBr, 0.5; K_2HPO_4, 0.2; NH_4 lactate, 0.6	0	0	0	0	0	0
607	16	0.011	KCl, 0.4; $Na_2HPO_4 \cdot 12H_2O$, 0.25; $(NH_4)_2$ oxalate, 0.6	2+	2c	1c	0	0	0
608	16	0.011	NaCl, 0.5; K_2HPO_4, 0.2; $(NH_4)_2SO_4$, 0.6; mannite, 0.5	0	0	0	0	0	0
610	16	0.011	NaCl, 0.5; K_2HPO_4, 0.2; $(NH_4)_2SO_4$, 0.6; maltose, 0.5	2+	4c	1c	0	1c	0
611	16	0.011	NaCl, 0.5; K_2HPO_4, 0.2; $(NH_4)_2SO_4$, 0.6; lactose, 0.5	2+	0	0	0	0	0
616	16	0.011	NaCl, 0.5; K_2HPO_4, 0.2; leucine, 0.5	3+	3+	3+	2+	2+	2+
620	16	0.006	NaCl, 0.5; K_2HPO_4, 0.2; tryosine, 0.25	3+	+	0	0	0	0
621	16	0.015	NaCl, 0.5; K_2HPO_4, 0.2; $(NH_4)_2SO_4$, 0.6; K_3 citrate, 0.6	3+	1c	0	0	0	0

[1] Cc. of NaOH (2.123M) per 1 cc. after sterilization = 0.003.

[2] Cc. of NaOH (2.123M) per 1 cc. after sterilization = 0.002.

TABLE 5

Capaldi-Proskauer media

NUMBER OF MEDIUM	BIBLIOGRAPHY	NaOH 2.123M: Cc. per 5 cc. before sterilization	PERCENTAGE COMPOSITION	RESULTS OF GROWTH: First transplant, Subcultures: 1	2	3	4	5	6	Second transplant, Subcultures: 1	2	3	4	5
			Basic mixture A: aspartic acid, 0.2; citric acid, 0.5; $CaCl_2$, 0.02; $MgSO_4 \cdot 7H_2O$, 0.2; KH_2PO_4, 0.2											
306	17	0.232	Mixture A plus dextrose, 0.2	3+	0	0	0	0	0	0	0	0	0	0
307[1]	17	0.22	Mixture A plus lactose, 0.2	3+	2+	3+	3+	0	0	0	0	0	0	0
308	17	0.215	Mixture A plus sucrose, 0.2	3+	3+	3+	+	0	0	0	0	0	0	0
309	17	0.23	Mixture A plus raffinose, 0.2	3+	3+	0	0	0	0	0	0	0	0	0
309a[2]	17	0.23	Mixture A	3+	3+	3+	0	4c	0	2+	2c	0	0	0
310	17	0.24	Mixture A plus maltose, 0.2	3+	2+	0	0	0	0	0	0	0	0	0
311[3]	17	0.23	Mixture A plus mannite, 0.2	0	0	0	0	0	0	0	0	0	0	0
			Basic mixture B: mannite, 0.2; $MgSO_4 \cdot 7H_2O$, 0.01; KH_2PO_4, 0.2; NaCl, 0.02											
314	17	0.04	Mixture B plus glycocoll, 0.2	3+	3+	3+	3+	2+	2+	4c	+	2c	+	0
315	17	0.035	Mixture B plus alanine, 0.2	3+	3+	2+	2+	2+	2+	+	+	3c	0	0
316	17	0.035	Mixture B plus leucine, 0.2	3+	3+	2+	2+	2+	3+	0	0	0	0	0
318	17	0.035	Mixture B plus tyrosine, 0.2	3+	3+	+	0	0	0	0	0	0	0	0
319	17	0.03	Mixture B plus urea, 0.2	0	0	0	0	0	0	0	0	0	0	0
321	17	0.03	Mixture B plus caffeine, 0.2	0	0	0	0	0	0	0	0	0	0	0
322	17	0.035	Mixture B plus glycocoll, 0.2; urea, 0.2	2+	2+	2+	2+	+	0	0	0	0	0	0
324	17	0.04	Mixture B plus NH_4 acetate, 0.2	0	0	0	0	0	0	0	0	0	0	0
326	17	0.04	Mixture B plus NH_4 lactate, 0.2	0	0	0	0	0	0	0	0	0	0	0
327	17	0.04	Mixture B plus $(NH_4)_2$ oxalate.H_2O, 0.2	+	0	0	0	0	0	0	0	0	0	0
328	17	0.08	Mixture B plus $(NH_4)_2$ tartrate, 0.2	2+	2+	0	0	0	0	0	0	0	0	0
329	17	0.06	Mixture B plus $(NH_4)_3$ citrate, 0.2	0	0	0	0	0	0	0	0	0	0	0
330	17	0.025	Mixture B plus $(NH_4)_2CO_3 \cdot H_2O$, 0.2	0	0	0	0	0	0	0	0	0	0	0
312	17	0.2	Aspartic acid, 0.2; dextrose, 0.2; $MgSO_4 \cdot 7H_2O$, 0.2; NaCl, 0.5; $Na_2PO_4 \cdot 12H_2O$, 0.2	3+	2+	+	0	0	0	2c	0	0	0	0
313[4]	17	0.065	Aspartic acid, 0.2; dextrose, 0.2; $CaCl_2$, 0.02; $MgSO_4 \cdot 7H_2O$, 0.01; KH_2PO_4, 0.02; NaCl, 0.02	3+	3+	3+	4c	0	0	3c	3c	0	0	0
331	17	0.02	NH_4NO_3, 0.2; mannite, 0.2; $MgSO_4 \cdot 7H_2O$, 0.01; KH_2PO_4, 0.02; NaCl, 0.02	0	0	0	0	0	0	0	0	0	0	0
332	17	0.02	$(NH_4)_2HPO_4$, 0.2; mannite, 0.2; $MgSO_4 \cdot 7H_2O$, 0.01; NaCl, 0.02	+	+	4c	0	0	0	0	0	0	0	0
333	17	0.04	Aspartic acid, 0.2; mannite, 0.2; $CaCl_2$, 0.02; $MgCl_2$, 0.01; KH_2PO_4, 0.2; NaCl, 0.02	3+	3+	3+	3+	3+	3+	+	+	+	+	+

[1] Cc. of NaOH (2.123 M) per 1 cc. after sterilization = 0.001.

[2] 309a: adaptation from 309.

[3] 311, 314–333 contain mannose in the original.

[4] Cc. of NaOH (2.123 M) per 1 cc. after sterilization = 0.002.

TABLE 6

Currie media

NUMBER OF MEDIUM	BIBLIOGRAPHY	NaOH 2.123M	PERCENTAGE COMPOSITION										RESULTS OF GROWTH					
													First transplant					
													Subcultures:					
		Cc. per 5 cc. before sterilization	NH_4NO_3	$NH_4H_2PO_4$	Aspartic acid	Sucrose	$FeSO_4 \cdot 7H_2O$	HCl w/5	$MgSO_4 \cdot 7H_2O$	KCl	KH_2PO_4	$NaNO_3$	1	2	3	4	5	6
142	20	0.02				5.0			0.02		0.1	0.2	0	0	0	0	0	0
143	20	0.036				3.0			0.02	0.02	0.2		0	0	0	0	0	0
144	20	0.04	0.25			15.0	0.002[1]	1.0	0.04[1]		0.2[1]		+	+	0	0	0	0
334	20	0.02				5.0			0.02		0.1	1.5[2]	0	0	0	0	0	0
335	20	0.03				5.0			0.02		0.1	0.2	0	0	0	0	0	0
336	20	0.02				5.0	0.001		0.05	0.05	0.1	0.3	0	0	0	0	0	0
337	20	0.02				10.0	0.001		0.05	0.05	0.1	0.3	0	0	0	0	0	0
338	20	0.02	0.25			15.0			0.025		0.1		+	0	0	0	0	0
339	20	0.04		0.2		5.0			0.04	0.02			3+	0	0	0	0	0
340	20	0.05		0.2		5.0	0.001		0.02	0.02			3+	+	0	0	0	0
341	20	0.05		0.2		5.0			0.02	0.02			3+	+	0	0	0	0
342	20	0.04		0.2		5.0	0.001		0.02	0.1			0	0	0	0	0	0
343	20	0.05		0.2		5.0			0.02		0.1		0	0	0	0	0	0
344	20	0.05		0.2		5.0	0.001		0.02	0.2			0	0	0	0	0	0
345	20	0.04		0.2		5.0			0.02	0.2			0	0	0	0	0	0
346[3]	20	0.035			0.2	5.0	0.001		0.02	0.02			2+	2+	+	0	2c	0
347[4]	20	0.03			0.2	5.0			0.02	0.02			+	0	+	0	0	0
348	20	0.04				5.0			0.02		0.1	0.25	0	0	0	0	0	0
349	20	0.02	0.12			5.0	0.001		0.02		0.1		0	0	0	0	0	0
350	20	0.02	0.12			5.0			0.02		0.1		0	0	0	0	0	0

[1] 144: Currie used one-half the quantities of $FeSO_4$, $MgSO_4$, and KH_2PO_4.

[2] 334: Currie used 0.15 $NaNO_3$.

[3] Cc. of NaOH (2.123M) per 1 cc. after sterilization = 0.002

[4] Cc. of NaOH (2.123M) per 1 cc. after sterilization = 0.003.

TABLE 7

Csapek media

NUMBER OF MEDIUM	BIBLIOGRAPHY	NaOH 2.123M		PERCENTAGE COMPOSITION														RESULTS OF GROWTH					
																		First transplant					
																		Subcultures:					
		Cc. per 5 cc. before sterilization	Cc. per 1 cc. after sterilization	Alanine	NH_4 acetate	NH_4 lactate	Aspartic acid	Betaine-HCl	Glycocoll	Leucine	Tyrosine	Urea	Sucrose	$FeSO_4 \cdot 7H_2O$	$MgSO_4 \cdot 7H_2O$	KCl	KH_2PO_4	1	2	3	4	5	6
442	21	0.03			1.0									0.001	0.05	0.05	0.1	0	0	0	0	0	0
443	21	0.035							1.0					0.001	0.05	0.05	0.1	3+	2+	0	0	0	0
444	21	0.02			1.0								3.0	0.001	0.05	0.05	0.1	0	0	0	0	0	0
445	21	0.038							1.0				3.0	0.001	0.05	0.05	0.1	3+	3+	2+	2+	2+	0
446	21	0.02	0.004		4.0								3.0	0.001	0.05	0.05	0.1	0	0	0	0	0	0
449	21	0.025		1.0									3.0	0.001	0.05	0.05	0.1	3+	3+	2+	+	2+	0
450	21	0.025	0.004		4.0									0.001	0.05	0.05	0.1	0	0	0	0	0	0
451	21	0.08			1.0								3.0	0.001	0.05	0.05	0.1	3+	3+	3+	2+	2+	2+
452	21	0.03								1.0			3.0	0.001	0.05	0.05	0.1	3+	+	+	0	0	0
453	21	0.02									1.0		3.0	0.001	0.05	0.05	0.1	0	0	0	0	0	0
454	21	0.04										4.0		0.001	0.05	0.05	0.1	0	0	0	0	0	0
455	21	0.002										1.0	3.0	0.001	0.05	0.05	0.1	0	0	0	0	0	0
457	21	0.17						1.0					3.0	0.001	0.05	0.05	0.1	0	0	0	0	0	0
458	21	0.105	0.025				1.0						3.0	0.001	0.05	0.05	0.1	2+	2+	+	0	0	0
460	21	0.14	0.01				1.0							0.001	0.05	0.05	0.1	3+	2+	+	0	0	0
10[1]	64												3.0	0.001	0.05	0.05		0	0	0	0	0	0

[1] 10: also contained K_2HPO_4, 0.1 per cent, and $NaNO_3$, 0.2 per cent. Tanner used $NaNO_3$ instead of KNO_3 in the original (22).

TABLE 8

Dolt media

NUMBER OF MEDIUM	BIBLIOGRAPHY	NaOH 2.123M		PERCENTAGE COMPOSITION	RESULTS OF GROWTH										
					First transplant						Second transplant				
					Subcultures:						Subcultures:				
		Cc. per 5 cc. before sterilization	Cc. per 1 cc. after sterilization		1	2	3	4	5	6	1	2	3	4	5
38	23	0.285	0.02	Aspartic acid, 2.5; $Na_2HPO_4 \cdot 12H_2O$, 0.5	3+	3+	3+	0	0	0	0	0	0	0	0
40	23			NH_4 lactate, 0.6; aspartic acid, 0.4; Na_2CO_3, 0.97; NaCl, 0.5; $Na_2HPO_4 \cdot 12H_2O$, 0.2	+	0	0	0	0	0	0	0	0	0	0
41	23			Aspartic acid, 1.0; Na_2CO_3, 2.6; NaCl, 0.5; $Na_2HPO_4 \cdot 12H_2O$, 0.2	0	0	0	0	0	0	0	0	0	0	0
60	23	0.186		Aspartic acid, 1.0; $Na_2HPO_4 \cdot 12H_2O$, 0.2	3+	0	0	0	0	0	0	0	0	0	0
69	23	0.43		Malic acid, 1.0; $(NH_4)_2HPO_4$, 0.2	0	0	0	0	0	0	0	0	0	0	0
196	23	0.015		Glycocoll, 1.0	4+	2+	2c	0	0	0	0	0	0	0	0
197	23	0.015		Glycocoll, 1.0; NaCl, 0.2	2+	0	0	0	0	0	0	0	0	0	0
198	23	0.005		Glycocoll, 1.0; KCl, 0.5[1]	0	0	0	0	0	0	0	0	0	0	0
199	23	0.015		Urea, 1.0	0	0	0	0	0	0	0	0	0	0	0
200	23	0.033		Aspartic acid, 1.0; NaCl, 0.2	0	0	0	0	0	0	0	0	0	0	0
201	23	0.17		Aspartic acid, 1.0; KCl, 0.2	3+	2+	2+	0	5c	0	0	0	0	0	0
202	23	0.175		Aspartic acid,1.0; NH_4Cl, 0.2	+	0	0	0	0	0	0	0	0	0	0
203	23	0.145	0.009	Aspartic acid, 1.0; $CaCl_2$, 0.2	2+	2+	0	0	0	0	0	0	0	0	0
204	23	0.168		Aspartic acid, 1.0; $MgCl_2$, 0.2	3+	2+	+	0	0	0	0	0	0	0	0
205	23	0.165		Aspartic acid, 1.0; Na_2SO_4, 0.2	3+	3+	0	0	0	0	3c	0	0	0	0
206	23	0.163		Aspartic acid, 1.0; K_2SO_4, 0.2	0	0	0	0	0	0	0	0	0	0	0
207	23	0.353		Aspartic acid, 1.0; $(NH_4)_2SO_4$, 0.2	4+	+	0	0	0	0	0	0	0	0	0
208	23	0.153	0.008	Aspartic acid, 1.0; $NaNO_3$, 0.2	4+	2+	0	0	0	0	0	0	0	0	0
209	23	0.158	0.002	Aspartic acid, 1.0; KNO_3, 0.2	4+	2+	0	0	0	0	0	0	0	0	0
210	23	0.18		Aspartic acid, 1.0; NH_4NO_3, 0.2	3+	2+	0	0	0	0	0	0	0	0	0
212	23	0.164		Aspartic acid, 1.0; K_2HPO_4, 0.2	4+	3+	2+	2+	+	0	0	0	0	0	0
213	23	0.167		Aspartic acid, 1.0; $(NH_4)_2HPO_4$, 0.2	3+	3+	2+	2+	2+	0	0	0	3c	4c	0
214	23	0.16		Aspartic acid, 1.0; $Na_2HPO_4 \cdot 12H_2O$, 0.4	4+	3+	+	+	+	0	4c	0	0	0	0
215	23	0.2		Aspartic acid, 1.0; $(NH_4)_2HPO_4$, 0.4	4+	4+	3+	3+	3+	2+	+	0	0	0	0
216	23	0.165		Aspartic acid, 1.0; $NaNO_3$,0.2; $Na_2HPO_4 \cdot 12H_2O$, 0.2	4+	+	0	0	0	0	0	0	0	0	0
217	23	0.169		Aspartic acid, 1.0; NH_4NO_3, 0.2; $(NH_4)_2HPO_4$, 0.2	3+	+	0	0	0	0	0	0	0	0	0
218	23	0.167		Aspartic acid, 1.0; $(NH_4)_2HPO_4$, 0.2; $NaNO_3$, 0.2	4+	3+	5c	0	0	0	0	0	0	0	0
219	23	0.165		Aspartic acid, 1.0; NH_4NO_3, 0.2; $NaNO_3$, 0.2	4+	2+	0	0	0	0	0	0	0	0	0
220	23	0.166		Aspartic acid, 1.0; $(NH_4)_2HPO_4$, 0.2; $Na_2HPO_4 \cdot 12H_2O$, 0.2	4+	3+	0	0	0	0	0	0	0	0	0
221	23			Glycocoll, 1.0; $(NH_4)_2HPO_4$, 0.2	0	0	0	0	0	0	0	0	0	0	0
222	23	0.057	0.003	NH_4 lactate, 1.0; $Na_2HPO_4 \cdot 12H_2O$, 0.2	0	0	0	0	0	0	0	0	0	0	0
223	23	0.295		Malic acid, 1.0; $(NH_4)_2HPO_4$, 0.2	0	0	0	0	0	0	0	0	0	0	0
224	23	0.246		$(NH_4)_2$ tartrate, 1.0; $Na_2HPO_4 \cdot 12H_2O$, 0.2	0	0	0	0	0	0	0	0	0	0	0
225	23		0.002	Glycocoll, 1.0; lactose, 1.0; $(NH_4)_2HPO_4$, 0.2	3+	+	0	0	0	0	0	0	0	0	0
226	23	0.055	0.002	NH_4 lactate, 1.0; lactose, 1.0; $Na_2HPO_4 \cdot 12H_2O$, 0.2	0	3c	4c	4c	0	0	0	0	0	0	0
228	23	0.17	0.002	Aspartic acid, 1.0; NH_4NO_3, 0.4	2+	2+	0	+	0	0	1c	+	0	0	0
229	23	0.158		Aspartic acid, 1.0; $NaNO_3$, 0.4	0	0	0	0	0	0	0	0	0	0	0
230	23	0.315	0.004	Aspartic acid.1.0	3+	2+	0	0	0	0	1c	0	0	0	0

[1] Dolt used only 0.2 per cent KCl.

TABLE 9

Fermi media

NUMBER OF MEDIUM	BIBLIOGRAPHY	NaOH 2.123M		PERCENTAGE COMPOSITION												RESULTS OF GROWTH					
																First transplant					
																Subcultures:					
		Cc. per 5 cc. before sterilization	Cc. per 1 cc. after sterilization	NH_4NO_3	$(NH_4)_2HPO_4$	$(NH_4)_2SO_4$	$(NH_4)_2$ tartrate	Glycerol	Lactose	Mannite	Salicin	Saponin	Sucrose	$Ca_3(PO_4)_2$	$MgSO_4 \cdot 7H_2O$	1	2	3	4	5	6
110[1]	24				1.0			4.5							0.02	2+	0	0	0	0 0	0
544	24	0.09			1.0								5.0	0.05	0.5	2+	0	0	0	0	0
545	24	0.03				1.0							5.0	0.05	0.5	0	0	0	0	0	0
546	24	0.05				1.0		5.0						0.05	0.5	0	0	0	0	0	0
547	24	0.035		1.0									5.0	0.05	0.5	0	0	0	0	0	0
548	24	0.04		1.0				5.0						0.05	0.5	0	0	0	0	0	0
549	24	0.2	0.01				1.0						5.0	0.05	0.5	3+	2+	2+	0	0	0
550	24	0.22	0.003				1.0	5.0						0.05	0.5	3+	2+	2+	2+	+	0
551	24	0.06			1.0					5.0				0.05	0.5	3+	2+	+	0	0	0
552	24	0.07			1.0				5.0					0.05	0.5	3+	0	0	0	0	0
553	24	0.065			1.0						1.0			0.05	0.5	2+	3c	0	0	0	0
554	24	0.14			1.0							1.0		0.05	0.5	3+	2+	2+	2+	0	0
555[2]	24	0.07			1.0			5.0						0.05	0.5	3+	2+	3+	3+	3+	2+

[1] 110: also contained KH_2PO_4, 0.1 per cent.

[2] Media 548 and 555 show 4c and + growths respectively in the first subculture of the second transplant.

TABLE 10

Frankel-Voges media

Number of medium	Bibliography	Percentage composition												Results of growth										
		NaOH 2.123M		NH_4 lactate	Aspartic acid	Dextrose	Lactose	Malic acid	Maltose	Sucrose	$CaCl_2$	$MgSO_4 \cdot 7H_2O$	K_2HPO_4	Na_2CO_3	NaCl	$Na_2HPO_4 \cdot 12H_2O$	First transplant						Second transplant				
		Cc. per 5 cc. before sterilization	Cc. per 1 cc. after sterilization														Subcultures:						Subcultures:				
																	1	2	3	4	5	6	1	2	3	4	5
510	27	0.27			1.0			0.7			0.001	0.2		0.25		0.2	3+	+	0		0	0	0	0	0	0	0
511	27	0.31	0.004		1.0			0.7		1.5	0.001	0.2		0.25		0.2	0	0	0		0	0	0	0	0	0	0
512	27	0.29	0.012		1.0		1.5	0.7			0.001	0.2		0.25		0.2	3+	3+	2+	2+	2+	+	+	3c	0	0	0
513	27	0.29	0.015		1.0			0.7	1.5		0.001	0.2		0.25		0.2	3+	3+	3+	0	0	0	2c	0	0	0	0
514	27	0.30	0.008		1.0	1.5		0.7			0.001	0.2		0.25		0.2	3+	3+	3+	0	0	0	4c	0	0	0	0
595	27	0.01		0.6	0.4								0.2		0.5		2+	2+	2+	2+	+	+	0	0	0	0	0

TABLE 11

Franzen-Egger media

NUMBER OF MEDIUM	BIBLIOGRAPHY	NaOH 2.123M	PERCENTAGE COMPOSITION													RESULTS OF GROWTH										
																First transplant						Second transplant				
																Subcultures:						Subcultures:				
		Cc. per 5 cc. before sterilization	Alanine	Aspartic acid	Glycocoll	Dextrose	Galactose	Lactose	Maltose	Sucrose	$CaCl_2$	$FeSO_4 \cdot 7H_2O$	$MgSO_4 \cdot 7H_2O$	KH_2PO_4	$NaCO_3$	1	2	3	4	5	6	1	2	3	4	5
235	28	0.15		1.2		0.54					0.021	0.0003	0.024	0.14	0.21	3+	3+	2+	+	+	0	+	1c	0	0	0
237[1]	28	0.13		1.2						0.54	0.021	0.0003	0.024	0.14	0.21	4+	4+	3+	3+	3+	3+	+	0	0	0	+
238	28	0.155		1.2			0.54				0.021	0.0003	0.024	0.14	0.21	4+	4+	3+	3+	2+	0	+	4c	0	2c	0
239	28	0.148		1.2				0.54			0.021	0.0003	0.024	0.14	0.21	4+	4+	3+	+	+	0	+	0	0	0	0
240	28	0.148		1.2					0.54		0.021	0.0003	0.024	0.14	0.21	4+	4+	3+	0	0	0	+	0	0	0	0
241	28				1.2	0.54					0.021	0.0003	0.024	0.14	0.21	4+	4+	0	0	0	0	+	0	0	0	0
243	28				1.2					0.54	0.021	0.0003	0.024	0.14	0.21	2+	+	+	0	0	0	0	3c	4c	0	0
244	28				1.2		0.54				0.021	0.0003	0.024	0.14	0.21	+	0	0	0	0	0	0	0	0	0	0
245	28				1.2			0.54			0.021	0.0003	0.024	0.14	0.21	3+	3+	2+	0	0	0	0	0	0	0	0
246	28				1.2				0.54		0.021	0.0003	0.024	0.14	0.21	3+	3+	3+	2+	+	0	3c	3c	2c	0	0
247	28		1.42			0.54					0.021	0.0003	0.024	0.14	0.21	3+	0	0	0	0	0	0	0	0	0	0
249	28		1.42							0.54	0.021	0.0003	0.024	0.14	0.21	3+	4+	3+	3+	3+	2+	+	0	0	0	0
250	28	•	1.42				0.54				0.021	0.0003	0.024	0.14	0.21	3+	2+	0	0	0	0	0	0	0	0	0
251	28		1.42					0.54			0.021	0.0003	0.024	0.14	0.21	3+	2+	2+	+	+	0	0	0	0	0	0
252	28		1.42						0.54		0.021	0.0003	0.024	0.14	0.21	4+	3+	3+	2+	2+	0	0	3c	0	0	0

[1] Cc. of NaOH (2.123 M) per 1 cc. after sterilization = 0.004.

TABLE 12

Galimard-Lacomme media

NUMBER OF MEDIUM	HELLIGRAFEY	NaOH 2.123M	PERCENTAGE COMPOSITION										RESULTS OF GROWTH										
													First transplant						Second transplant				
													Subcultures:						Subcultures:				
		Cc. per 5 cc. before sterilization	Aspartic acid	Glycocoll	Leucine	Tyrosine	Urea	Ca-glycero-PO_4	Glycerol	$MgSO_4 \cdot 7H_2O$	$KHCO_3$	NaCl	1	2	3	4	5	6	1	2	3	4	5
70[1]	23				0.75	Trace		0.02	1.5	0.05		0.005	+	0	0	0	0	0	0	0	0	0	0
146	31							0.2	1.5	0.05	0.03	0.5	0	0	0	0	0	0	0	0	0	0	0
526	31	0.006		1.0				0.2	1.5	0.05	0.03	0.5	2+	2+	2+	0	0	0	0	0	0	0	0
527	31				1.0[2]			0.2	1.5	0.05	0.03	0.5	3+	3+	3+	0	0	0	2+	3+	0	0	0
528	31	0.004				1.0		0.2	1.5	0.05	0.03	0.5	3+	0	0	0	0	0	0	0	0	0	0
529[3]	31	0.09	1.0					0.2	1.5	0.05	0.03	0.5	0	0	0	0	0	0	0	0	0	0	0
530	31						1.0	0.2	1.5	0.05	0.03	0.5	0	0	0	0	0	0	0	0	0	0	0
531	31	0.004			0.1	0.9		0.2	1.5	0.05	0.03	0.5	+	0	0	0	0	0	0	0	0	0	0
532	31	0.003		0.5			0.5	0.2	1.5	0.05	0.03	0.5	2+	+	0	0	0	0	0	0	0	0	0
76[4]	44							0.2		0.05	0.1	0.5	0	0	0	0	0	0	0	0	0	0	0

[1] Dolt's adaptation from Galimard and Lacomme; alanine (0.2 per cent) and Na_2CO_3 (0.024 per cent) were also included.

[2] Natural leucine was used.

[3] Cc. of NaOH (2.123 M) per 1 cc. after sterilization = 0.015.

[4] Lepierre also used a glycoprotein.

TABLE 13

Gordon media

NUMBER OF MEDIUM	BIBLIOGRAPHY	NaOH 2.123M		PERCENTAGE COMPOSITION													RESULTS OF GROWTH					
		Cc. per 5 cc. before sterilisation	Cc. per 1 cc. after sterilisation	Alanine	$(NH_4)_2CO_3 \cdot H_2O$	NH_4Cl	$(NH_4)_2HPO_4$	Aspartic acid	Glycocoll	Urea	Dextrose	$CaCl_2$	$MgSO_4 \cdot 7H_2O$	K_2HPO_4	NaCl	First transplant					
																Subcultures:					
																1	2	3	4	5	6
579	33	0.01							1.0		1.0	0.01	0.2	0.1	0.5	0	0	0	0	0	0
580	33	0.01							1.0			0.01	0.2	0.1	0.5	3+	3+	3+	3+	3+	0
581	33	0.02	0.003			1.0					1.0	0.01	0.2	0.1	0.5	0	0	0	0	0	0
582	33	0.02	0.002			1.0						0.01	0.2	0.1	0.5	0	0	0	0	0	0
583	33	0.04					1.0				1.0	0.01	0.2	0.1	0.5	3+	0	0	0	0	0
584	33	0.03					1.0					0.01	0.2	0.1	0.5	0	0	0	0	0	0
585	33				1.0						1.0	0.01	0.2	0.1	0.5	0	0	0	0	0	0
586	33				1.0							0.01	0.2	0.1	0.5	0	0	0	0	0	0
587[1]	33	0.01									1.0	0.01	0.2	0.1	0.5	0	0	0	0	0	0
588[1]	33	0.01										0.01	0.2	0.1	0.5	0	0	0	0	0	0
589	33									1.0	1.0	0.01	0.2	0.1	0.5	0	0	0	0	0	0
590	33	0.005									1.0	0.01	0.2	0.1	0.5	0	0	0	0	0	0
591	33	0.08	0.008					1.0			1.0	0.01	0.2	0.1	0.5	0	0	0	0	0	0
592	33	0.12	0.012					1.0				0.01	0.2	0.1	0.5	0	0	0	0	0	0
593	33	0.005		1.0							1.0	0.01	0.2	0.1	0.5	3+	0	0	0	0	0
594	33	0.005		1.0								0.01	0.2	0.1	0.5	0	0	0	0	0	0
94[2]	33	0.006				0.1						0.01	0.2	0.1	0.5	0	0	0	0	0	0
95[2]	33	0.001			0.1							0.01	0.2	0.1	0.5	0	1c	0	0	0	0
96[2]	33	0.002						0.1				0.01	0.2	0.1	0.5	3+	+	0	0	0	0
97[2]	33	0.006					0.1					0.01	0.2	0.1	0.5	0	0	0	0	0	0
98[2]	33	0.004		0.1								0.01	0.2	0.1	0.5	0	0	0	0	0	0
99[2]	33	0.006									0.1	0.01	0.2	0.1	0.5	0	0	0	0	0	0

[1] 587 and 588 also contained KNO_3, 1.0 per cent.

[2] 94, 95, 96, 97, 98, and 99: Adaptations from Gordon. The N-compounds were used in one tenth the original amounts.

TABLE 14

Hadley media

NUMBER OF MEDIUM	BIBLIOGRAPHY	NaOH 2.123M		PERCENTAGE COMPOSITION												RESULTS OF GROWTH										
																First transplant						Second transplant				
																Subcultures:						Subcultures:				
		Cc. per 5 cc. before sterilization	Cc. per 1 cc. after sterilization	NH_4 lactate	$(NH_4)_2HPO_4$	Aspartic acid	Glycocoll	Urea	Glycerol	$CaCl_2$	$MgSO_4 \cdot 7H_2O$	KNO_3	K_2HPO_4	NaCl		1	2	3	4	5	6	1	2	3	4	5
128	34	0.017		0.05	1.0	1.0			4.0	0.01	0.03	0.03	0.3			3+	3+	3+	3+	2+	+	0	0	0	0	0
129	34	0.214	0.008	0.08		1.5			2.5			0.03	0.1	0.1		0	0	0	0	0	0	0	0	0	0	0
130	34	0.165			1.0	1.0			4.0	0.01		0.3[1]	0.3	0.6		3+	0	0	0	0	0	0	0	0	0	0
131	34	0.06		0.75			0.1		3.4	0.08	0.32		0.23	0.6		3+	0	0	0	0	0	0	0	0	0	0
132	34	0.06		0.75			0.1		3.0	0.08	0.32		0.23	0.6		3+	3+	3+	3+	2+	+	0	0	0	0	0
133	34	0.1	0.012		1.75	1.0			4.0	0.01			0.03	0.6		2+	0	5c	+	0	0	0	0	0	0	0
134	34	0.18		3.25			0.5	0.25	17.0	0.04	0.16		1.25	3.0		3+	2+	0	0	0	0	0	0	0	0	0
135	34	0.202		3.2				1.6	15.0	0.05	0.15		1.25	3.0		0	0	0	0	0	0	0	0	0	0	0
231[2]	34	0.18		0.5	1.0	1.0			4.0		0.03		0.3	0.1		4+	4+	4+	4+	4c	3+	+	3c	4c	3c	0

[1] 130: only 0.03 per cent KNO_3 was used in the original.

[2] 231: also contained $Fe_3(PO_4)_2 \cdot 8H_2O$, 0.02 per cent.

TABLE 15

Koessler-Hanke media

NUMBER OF MEDIUM	BIBLIOGRAPHY	NaOH 2.123M	PERCENTAGE COMPOSITION											RESULTS OF GROWTH										
							Salt mixture							First transplant						Second transplant				
														Subcultures:						Subcultures:				
		Cc. per 5 cc. before sterilization	NH_4Cl	Histidine-HCl	Glycerol	KNO_3	KCl	$CaCl_2$	KH_2PO_4	$NaHCO_3$	$NaSO_4$	NaCl		1	2	3	4	5	6	1	2	3	4	5
119	40	0.024		0.1			0.0075	0.005	0.2	0.2	0.01	0.75		+	0	0	0	0	0	0	0	0	0	0	0
120	40	0.02	0.1	0.1			0.0075	0.005	0.2	0.2	0.01	0.75		4+	4+	+	2+	+	4c	+	+	+	3c	4c
121	40	0.022		0.1	2.0		0.0075	0.005	0.2	0.2	0.01	0.75		3+	3+	3+	3+	+	+	2c	3c	2c	0	0
122	40	0.019	0.1	0.1	2.0		0.0075	0.005	0.2	0.2	0.01	0.75		3+	4+	4+	4+	4+	4+	+	4c	3c	2c	3c
123	40	0.019		0.1		0.05	0.0075	0.005	0.2	0.2	0.01	0.75		0	0	0	0	0	0	0	0	0	0	0
124	40	0.02	0.1	0.1		0.05	0.0075	0.005	0.2	0.2	0.01	0.75		3+	3+	3+	2+	2+	2+	+	+	+	+	+
125	40	0.019		0.1	2.0	0.05	0.0075	0.005	0.2	0.2	0.01	0.75		2+	2+	3+	2+	2+	0	1c	0	0	0	0
126	40	0.031	0.1	0.1	2.0	0.05	0.0075	0.005	0.2	0.2	0.01	0.75		4+	4+	3+	4+	4+	4+	+	5c	+	0	+
127[1]	40	0.026	0.1	0.1		0.05	0.0075	0.005	0.2	0.2	0.01	0.75		0	0	0	0	0	0	0	0	0	0	0

[1] 127: also contained dextrose, 2.5 per cent.

TABLE 16

Lockemann media

NUMBER OF MEDIUM	BIBLIOGRAPHY	NaOH 2.123M	PERCENTAGE COMPOSITION														RESULTS OF GROWTH										
																	First tranplant						Second transplant				
																	Subcultures:						Subcultures:				
		Cc. per 5 cc. before sterilization	$(NH_4)_2HPO_4$	Aspartic acid	$CaH_2(PO_4)_2 \cdot 2H_2O$	M citric acid	Glycerol	Mg_3 citrate	$MgHPO_4 \cdot 3H_2O$	$MgSO_4 \cdot 7H_2O$	M/3 H_3PO_4	K_2HPO_4	KH_2PO_4	$NaHPO_4 \cdot 12H_2O$	$NaH_2PO_4 \cdot 4H_2O$	M/2 H_2SO_4	1	2	3	4	5	6	1	2	3	4	5
100	45	0.215		0.529		1.0	2.0		0.493		1.0	0.018		0.036		1.0	4+	4+	4+	4+	4+	4+	2+	+	+	+	+
101	45	0.214		0.5			2.0	0.25		0.06			0.4		0.3		3+	4+	4+	4+	3+	3+	0	0	0	0	0
102	45	0.213		0.529		1.0	2.0		0.493		1.0[1]	0.035				1.0	4+	4+	4+	3+	3+	4+	0	3c	+	+	5c
285	45		0.6				4.0			0.6							0	0	0	0	0	0	0	0	0	0	0
286	45	0.08	0.6				4.0			0.6					0.3		4+	4+	4+	2+	3+	2+	2c	3c	2c	6c	0
287	45	0.145					2.0	0.25		0.1[2]			0.4		0.3		4+	2+	3+	3+	3+	2+	0	0	0	0	0
289	45	0.2		0.5		1.2	2.0		0.4		2.5		0.025	0.025			4+	4+	4+	3+	4+	4+	+	2+	+	+	+
290[3]	45	0.24		0.5	0.025	1.2	2.0		3.75		2.5		0.025	0.025			4+	4+	4+	4+	4+	0	+	5c	3c	0	0
291	45	0.21		0.5	0.025	1.2	2.0		0.375		2.5						4+	4+	4+	4+	4+	0	+	+	+	+	0
292	45	0.275		0.5	0.29	1.2	2.0				2.5		0.025	0.025			4+	3+	4+	4+	4+	+	+	0	0	0	0
293	45	0.185		0.529		1.0	2.0		0.493		2.0			0.072		1.0	4+	4+	4+	4+	4+	+	2+	2+	2+	+	0
284[4]	45		0.6				4.0										0	0	0	0	0	0	0	0	0	0	0

[1] 102: Lockemann used 2.0 per cent M H_3PO_4.

[2] 287: Original contained no $MgSO_4$; medium here and in the original also contained 0.5 per cent $(NH_4)_2SO_4$.

[3] 290: Cc. of NaOH (2.123 M) per 1 cc. after sterilization = 004. Original used only 0.375 per cent $MgHPO_4$.

[4] 284: Lowenstein's medium (47).

TABLE 17

Maassen media

NUMBER OF MEDIUM	BIBLIOGRAPHY	NaOH 2.123M		PERCENTAGE COMPOSITION								RESULTS OF GROWTH										
												First transplant						Second transplant				
												Subcultures:						Subcultures:				
		Cc. per 5 cc. before sterilization	Cc. per 1 cc. after sterilization	Aspartic acid	Lactic acid	Malic acid	Tartaric acid	$CaCl_2$	$MgSO_4 \cdot 7H_2O$	$NaCO_3$	$Na_2HPO_4 \cdot 12H_2O$	1	2	3	4	5	6	1	2	3	4	5
461	48	0.263	0.006	1.0		0.7		0.001	0.04	0.25	0.2	3+	2+	0	0	0	0	0	0	0	0	0
515	48	0.21	0.02	1.0	0.7			0.001	0.04	0.25	0.2	3+	3+	2+	+	0	0	0	0	0	0	0
516	48	0.24	0.018	1.0			0.7	0.001	0.4	0.25	0.2	3+	2+	2+	+	0	0	0	0	0	0	0
517[1]	48	0.3	0.01	1.0				0.001	0.4	0.25	0.2	3+	2+	2+	0	0	0	+	0	0	0	0
518[2]	48	0.36		1.0		0.7		0.001	0.4			+	0	0	0	0	0	0	0	0	0	0
519[3]	48	0.27				0.7		0.001	0.4	0.25	0.2	0	0	0	0	0	0	0	0	0	0	0

[1] 517: also contained citric acid, 0.7 per cent.

[2] 518: also contained K_2CO_3, 0.25 per cent, and K_2HPO_4, 0.2 per cent.

[3] 519: also contained urea, 1.0 per cent.

TABLE 18

Naegeli media

NUMBER OF MEDIUM	BIBLIOGRAPHY	NaOH 2.123M: Cc. per 5 cc. before sterilization	NaOH 2.123M: Cc. per 1 cc. after sterilization	PERCENTAGE COMPOSITION	RESULTS OF GROWTH: First transplant, Subcultures: 1	2	3	4	5	6	Second transplant, Subcultures: 1	2	3	4	5
13a	51	0.23	0.003	$(NH_4)_2$ tartrate, 1.0; $CaCl_2$, 0.01; $MgSO_4 \cdot 7H_2O$, 0.02; K_2HPO_4, 0.1	+	0	0	0	0	0	0	0	0	0	0
165[1]	51	0.35		$(NH_4)_2$ tartrate, 1.0; histidine-HCl, 1.0; $CaCl_2$, 0.01; $MgSO_4 \cdot 7H_2O$, 0.2; K_2HPO_4, 0.1	4+	3+	2+	+	+	2+	2c	0	0	3c	2c
556	51	0.12	0.003	$(NH_4)_2SO_4$, 0.006; $(NH_4)_2$ tartrate, 0.5; sucrose, 10.0; $CaCl_2$, 0.0015; $MgSO_4 \cdot 7H_2O$, 0.006; K_2HPO_4, 0.035	+	0	0	0	0	0	0	0	0	0	0
557	51	0.2	0.008	$(NH_4)_2$ tartrate, 1.0; sucrose, 10.0; $CaCl_2$, 0.0025; $MgSO_4 \cdot 7H_2O$, 0.01; K_2HPO_4, 0.5	3+	+	+	0	0	0	0	0	0	0	0
558	51			K_2 tartrate $\cdot \frac{1}{2} H_2O$, 2.5; $CaCl_2$, 0.01; $MgSO_4 \cdot 7H_2O$, 0.04; K_2HPO_4, 0.5; $NaNO_3$, 1.0	0	0	0	0	0	0	0	0	0	0	0
559	51	0.4		NH_4NO_3, 0.2; glycerol, 7.5; $CaCl_2$, 0.005; $MgSO_4 \cdot 7H_2O$, 0.15[2]; K_2HPO_4, 0.2	3+	2+	2+	0	0	0	0	0	0	0	0
560	51			Urea, 0.2; glycerol, 7.5; $CaCl_2$, 0.005; $MgSO_4 \cdot 7H_2O$, 0.015; K_2HPO_4, 0.2	+	+	0	+	0	0	0	0	0	0	0
561	51			Sucrose, 3.0; $CaCl_2$, 0.008; $MgSO_4 \cdot 7H_2O$, 0.032; $K_4Fe(CN)_6 \cdot 3H_2O$, 0.6; K_2HPO_4, 0.1	3+	0	0	0	0	0	0	0	0	0	0
562	51			Sucrose, 10.0; $CaCl_2$, 0.006; $MgSO_4 \cdot 7H_2O$, 0.026; $K_4Fe(CN)_6 \cdot 3H_2O$, 1.0; K_2HPO_4, 0.1	3+	2+	2+	0	0	0	0	0	0	0	0
563	51	0.08	0.002	Aspartic acid, 0.5; glycerol, 3.0; $CaCl_2$, 0.02, $MgCl_2$, 0.05; K_2HPO_4, 0.2	3+	3+	2+	2+	3+	3+	+	0	3c	0	0
564	51	0.06	0.008	$(NH_4)_2$ HPO_4, 0.1; aspartic, 0.5; glycerol, 1.0; $MgCl_2$, 0.1; KNO_3, 0.1	3+	3+	3+	3+	3+	3+	+	+	0	0	0
565	51	0.24	0.005	$(NH_4)_2$ tartrate, 1.0; $CaCl_2$, 0.0055; $MgSO_4 \cdot 7H_2O$, 0.016; K_2HPO_4, 0.103; K_2SO_4, 0.013	3+	0	0	0	0	0	0	0	0	0	0
566	51			$(NH_4)_2SO_4$, 0.017; $CaCl_2$, 0.0055; $MgSO_4 \cdot 7H_2O$, 0.016; K_2HPO_4, 0.1	0	0	0	0	0	0	0	0	0	0	0
567	51			$CaCl_2$, 0.01; $MgSO_4 \cdot 7H_2O$, 0.02; K_2HPO_4, 0.1	0	0	0	0	0	0	0	0	0	0	0
568	51	0.36		$(NH_4)_2$ tartrate, 0.5; citric acid, 0.7; sucrose, 10.0; K_2HPO_4, 0.1	3+	0	+	0	0	0	0	0	0	0	0
569	51	0.33	0.004	$(NH_4)_2$ tartrate, 0.5; citric acid, 0.7; sucrose, 10.0; K_2HPO_4, 0.5	3+	0	0	0	0	0	0	0	0	0	0

[1] Adaptation from Naegeli by Mellanby and Twort (50).

[2] Original of 559 used 0.015 per cent $MgSO_4$.

TABLE 18—*Continued*

Naegeli media—Continued

NUMBER OF MEDIUM	BIBLIOGRAPHY	NaOH 2.123M		PERCENTAGE COMPOSITION	RESULTS OF GROWTH										
					First transplant						Second transplant				
					Subcultures:						Subcultures:				
		Cc. per 5 cc. before sterilization	Cc. per 1 cc. after sterilization		1	2	3	4	5	6	1	2	3	4	5
571	51	0.02		NH_4 acetate, 1.0; $CaCl_2$, 0.01; $MgSO_4 \cdot 7H_2O$, 0.02; K_2HPO_4, 0.1	3+	0	0	0	0	0	+	0	0	0	0
572	51	0.07		NH_4 lactate, 1.0; $CaCl_2$, 0.01; $MgSO_4 \cdot 7H_2O$, 0.02; K_2HPO_4, 0.1	0	0	0	0	0	0	+	0	0	0	0
573	51	0.11		$(NH_4)_3$ citrate, 1.0; $CaCl_2$, 0.01; $MgSO_4 \cdot 7H_2O$, 0.02; K_2HPO_4, 0.1	3+	+	0	0	0	0	0	0	0	0	0
574	51	0.118	0.013	Aspartic acid, 1.0; $CaCl_2$, 0.01; $MgSO_4 \cdot 7H_2O$, 0.02; K_2HPO_4, 0.1	0	0	0	0	0	0	0	0	0	0	0
575	51	0.115		Leucine, 1.0; $CaCl_2$, 0.01; $MgSO_4 \cdot 7H_2O$, 0.02; K_2HPO_4, 0.1	3+	2+	3+	3+	3+	0	0	4c	4c	0	0
576	51	0.11		$(NH_4)_2HPO_4$, 1.42; $(NH_4)_2SO_4$, 0.142; NH_4H tartrate, 0.71; sucrose, 8.5; $CaCl_2$, 0.042; $MgSO_4 7H_2O$, 0.11[a]	3+	2+	2+	2+	2+	2+	0	0	0	0	0
577	51	0.4		CH_3COOH, 0.8; NH_4 acetate, 1.0; $(NH_4)_2$ HPO_4, 0.4; $(NH_4)_2SO_4$, 0.02; glycerol, 4.0; $CaCl_2$, 0.006; $MgSO_4 \cdot 7H_2O$, 0.016	+	0	0	0	0	0	0	0	0	0	0
578	51	0.12		$(NH_4)_2SO_4$, 0.006; $(NH_4)_2$ tartrate, 0.5; sucrose, 10.0; $CaCl_2$, 0.0015; $MgSO_4 \cdot 7H_2O$, 0.006; K_2HPO_4, 1.0	2+	+	0	0	0	0	0	0	0	0	0
623	51	0.283		$(NH_4)_2$ tartrate, 1.0; $CaCl_2$, 0.02; $MgSO_4 \cdot 7H_2O$, 0.04; K_2HPO_4, 0.2; sucrose, 3.0	1c	0	0	0	0	0	0	0	0	0	0
624	51	0.562		$(NH_4)_2$ HPO_4, 0.64; $(NH_4)_2SO_4$, 0.016; $(NH_4)_2$ tartrate, 0.8; $CaCl_2$, 0.008; $MgSO_4 \cdot 7H_2O$, 0.016; sucrose, 0.8; tartaric acid, 0.8	2+	1c	0	0	0	0	0	0	0	0	0
570	51	0.38		$(NH_4)_2$ tartrate, 0.5; citric acid, 0.7; sucrose, 10.0; K_2HPO_4, 5.0	3+	+	0	0	0	0	0	0	0	0	0

[a] 576: original used 0.011 per cent $MgSO_4$.

TABLE 19

Omelianski-Winogradsky series[1]

NUMBER OF MEDIUM	BIBLIOGRAPHY	NaOH 2.123M		PERCENTAGE COMPOSITION
		Cc. per 5 cc. before sterilization	Cc. per 1 cc. after sterilization	
506	54			$(NH_4)_2SO_4$, 0.1; $MgSO_4 \cdot 7H_2O$, 0.05; K_2PO_4, 0.1; NaCl, trace.
507	54			$(NH_4)_2HPO_4$, 0.1; $MgSO_4 \cdot 7H_2O$, 0.05; K_2PO_4, 0.1; NaCl, trace.
508	54	0.05	0.008	Aspartic acid, 0.5; $MgSO_4 \cdot 7H_2O$, 0.05; K_2PO_4, 0.1; NaCl, trace.
539	53			$(NH_4)_2SO_4$, 0.2; $FeSO_4 \cdot 7H_2O$, 0.04; $MgCO_3$, 1.0; $MgSO_4 \cdot 7H_2O$, 0.05; K_2PO_4, 0.1; NaCl, 0.2
540	53			$(NH_4)_2SO_4$, 0.3; $MgSO_4 \cdot 7H_2O$, 0.05; K_2PO_4, 0.1
541	53	0.05	0.005	$(NH_4)_2SO_4$, 0.3; $FeSO_4 \cdot 7H_2O$, 2.0; $MgSO_4 \cdot 7H_2O$, 0.05; K_2PO_4, 0.1
542	53			$FeSO_4 \cdot 7H_2O$, 0.4; $MgSO_4 \cdot 7H_2O$, 0.3; K_2PO_4, 0.5; Na_2CO_3, 1.0; NaCl, 0.5; $NaNO_3$, 1.0
8[2]	64			$(NH_4)_2SO_4$, 0.1; $FeSO_4 \cdot 7H_2O$, 0.04; $MgSO_4 \cdot 7H_2O$, 0.05; NaCl, 0.2; KH_2PO_4, 0.1; 4 $MgCO_3$, $Mg(OH)_2 \cdot 5H_2O$, 0.5
533	82			K_2PO_4, 0.05; Na_2CO_3, 0.1; $NaNO_2$, 0.2
534	82			$MgSO_4 \cdot 7H_2O$, 0.03; K_2PO_4, 0.05; Na_2CO_3, 0.05; NaCl, 0.05; $NaNO_2$, 0.1
7[2]	64			$(NH_4)_2SO_4$, 0.1; $FeSO_4 \cdot 7H_2O$, 0.04; $MgCO_3$, 0.5; $MgSO_4 \cdot 7H_2O$, 0.05; NaCl, 0.2; K_2HPO_4, 0.1
535	78			$(NH_4)_2SO_4$, 0.2; $MgSO_4 \cdot 7H_2O$, 0.05; K_2PO_4, 0.1; Na_2CO_3, 0.5; $CaCl_2$, trace
536	78			$(NH_4)_2SO_4$, 0.4; $MgSO_4 \cdot 7H_2O$, 0.05; K_2PO_4, 0.1; Na_2CO_3, 0.6; $CaCl_2$, trace
537	79			$(NH_4)SO_4$, 2.0; $MgCO_3$, 0.02; $MgSO_4 \cdot 7H_2O$, 0.05; K_2PO_4, 0.1; $CaCl_2$, trace
538	78			$(NH_4)_2SO_4$, 0.1; $MgCO_3$, 0.5; K_2PO_4, 0.1
543	81			K_2PO_4, trace; Na_2CO_3, 0.1; $NaNO_3$, 0.2
597	80			$FeSO_4 \cdot 7H_2O$, 0.002; $MgSO_4 \cdot 7H_2O$, 0.05; K_2PO_4, 0.1; NaCl, 0.002; $MnSO_4 \cdot 4H_2O$, 0.002; dextrose, 3.0; $NaHCO_3$, 0.1064

[1] All showed no growth except 508, 8, 535 and 536, in which the growth was very slight for the first subculture of the first transplant.

[2] 7 and 8: adaptations from Winogradsky.

TABLE 20

Pieper-Humphrey-Acree media

NUMBER OF MEDIUM	BIBLIOGRAPHY	NaOH 2.123M: Cc. per 5 cc. before sterilization	NaOH 2.123M: Cc. per 1 cc. after sterilization	PERCENTAGE COMPOSITION	RESULTS OF GROWTH: First transplant: Subcultures: 1	2	3	4	5	6	Second transplant: Subcultures: 1	2	3	4	5
				Basic mixture: K_2HPO_4, 0.4; $MgSO_4 \cdot 7H_2O$, 0.2											
30	56	0.05	0.003	Basic mixture plus $(NH_4)_2HPO_4$, 0.2	2c	0	0	0	0	0	0	0	0	0	0
31	56	0.028	0.006	Basic mixture plus glucosamine-HCl, 0.4; sucrose, 2.0	4+	+	0	0	0	0	0	0	0	0	0
32	56			Basic mixture plus glycocoll, 0.4; sucrose, 4.0	3+	3+	3+	0	0	0	+	+	0	0	0
33	56	0.075		Basic mixture plus $(NH_4)_2HPO_4$, 0.4; aspartic acid, 0.4; glycerol, 4.0; sucrose, 4.0	3+	3+	0	0	0	0	+	0	0	0	0
34	56	0.06		Basic mixture plus betaine-HCl, 0.4; lactose, 3.2	2+	0	0	0	0	0	0	0	0	0	0
35	56		0.004	Basic mixture plus leucine, 0.4; dextrose, 2.5	2+	0	0	0	0	0	0	0	0	0	0
37	56	0.08		Aspartic acid, 0.4; $MgSO_4 \cdot 7H_2O$, 0.2; sucrose, 4.0	2+	0	0	0	0	0	0	0	0	0	0
630	56	0.033		Basic mixture plus $(NH_4)_2HPO_4$, 0.2; lactose, 2.0	3+	3+	0	0	0	0	3c	0	0	0	0
631	56	0.028		Basic mixture plus $(NH_4)_2HPO_4$, 0.2; maltose, 2.0	3+	3+	3c	0	0	0	0	0	0	0	0
632	56	0.028		Basic mixture plus $(NH_4)_2HPO_4$, 0.2; sucrose, 2.0	0	0	0	0	0	0	0	0	0	0	0
633	56	0.033		Basic mixture plus $(NH_4)_2HPO_4$, 0.2; galactose, 2.0	3+	2+	0	0	0	0	0	0	0	0	0
634	56	0.033		Basic mixture plus $(NH_4)_2HPO_4$, 0.2; dextrose, 2.0	4+	3+	0	0	0	0	2c	0	0	0	0
635	56	0.167		Basic mixture plus $(NH_4)_2HPO_4$, 0.2; glucosamine-HCl, 2.0	0	0	0	0	0	0	0	0	0	0	0
636	56	0.042		Basic mixture plus aspartic acid, 0.2; sucrose, 4.0	4+	4+	3+	0	2+	0	2c	6c	0	2c	0
637	56	0.005		Basic mixture plus sucrose, 4.0; caffeine, 0.2	3+	2+	3c	0	0	0	1c	0	0	0	0
638	56	0.01		Basic mixture plus leucine, 0.2; sucrose, 4.0	3+	3+	3+	2+	4c	0	2c	0	0	0	0
639	56	0.01		Basic mixture plus sucrose, 4.0; creatinine, 0.2	3+	3+	0	0	0	0	0	0	0	0	0
640	56	0.042		Basic mixture plus betaine-HCl, 0.2; sucrose, 4.0	0	0	0	0	0	0	0	0	0	0	0
641	56	0.106		Basic mixture plus $(NH_4)_2HPO_4$, 0.4; aspartic acid, 0.4; glycerol, 4.0; galactose, 4.0	0	0	0	0	0	0	0	0	0	0	0
642	56	0.106		Basic mixture plus $(NH_4)_2HPO_4$, 0.4; aspartic acid, 0.4; glycerol, 4.0; lactose, 4.0	3+	3+	3+	3+	3+	0	2c	3c	0	0	0
643	56	0.033		Basic mixture plus sucrose, 4.0; creatinine, 0.4	0	0	0	0	0	0	0	0	0	0	0
644	56	0.033		Basic mixture plus sucrose, 4.0; caffeine, 0.4	0	0	0	0	0	0	0	0	0	0	0
645	56	0.095		Basic mixture plus aspartic acid, 0.2; dextrose 4.0	3+	0	0	0	0	0	0	0	0	0	0

TABLE 21
Proskauer-Beck series

NUMBER OF MEDIUM	BIBLIOGRAPHY	NaOH 2.123M		PERCENTAGE COMPOSITION		RESULTS OF GROWTH										
						First transplant						Second transplant				
						Subcultures:						Subcultures:				
		Cc. per 5 cc. before sterilization	Cc. per 1 cc. after sterilization	C and N substances	Inorganic substances	1	2	3	4	5	6	1	2	3	4	5
358	57	0.055		Aspartic acid, 0.5; citric acid, 0.075; glucose, 1.0; glycerol, 4.0	K_2PO_4, 0.5; $MgSO_4 \cdot 7H_2O$, 0.25; K_2SO_4, 0.25; NaCl, 0.15	3+	0	0	0	0	0	0	0	0	0	0
359	57			Aspartic acid, 0.5; citric acid, 0.075; glycerol, 4.0	K_2PO_4, 0.5; $MgSO_4 \cdot 7H_2O$, 0.25; K_2SO_4, 0.25; NaCl, 0.15	4+	4+	4+	4+	3+	3+	+	+	5c	0	+
360	57	0.036		Aspartic acid, 0.5; glucose, 1.0; glycerol, 4.0	K_2PO_4, 0.5; $MgSO_4 \cdot 7H_2O$, 0.25; K_2SO_4, 0.25; NaCl, 0.15	4+	4+	0	0	0	0	+	+	+	0	0
361	57	0.03	0.003	Aspartic acid, 0.5; glycerol, 4.0	K_2PO_4, 0.5; $MgSO_4 \cdot 7H_2O$, 0.25; K_2SO_4, 0.25; NaCl, 0.15	4+	4+	3+	3+	2+	2+	+	4c	0	0	+
362	57	0.04		Aspartic acid, 0.5; glycerol, 1.5	K_2PO_4, 0.5; $MgSO_4 \cdot 7H_2O$, 0.25; K_2SO_4, 0.25; NaCl, 0.15	3+	3+	3+	3+	2+	2+	+	4c	0	0	+
363	57	0.05	0.003	Aspartic acid, 0.5; citric acid, 0.075; glucose, 1.0	K_2PO_4, 0.5; $MgSO_4 \cdot 7H_2O$, 0.25; K_2SO_4, 0.25; NaCl, 0.15	3+	3+	+	4c	0	0	+	0	0	0	0
364	57	0.025	0.003	Aspartic acid, 0.5; glycerol, 1.0	K_2PO_4, 0.5; $MgSO_4 \cdot 7H_2O$, 0.25; K_2SO_4, 0.25; NaCl, 0.15	3+	3+	+	+	+	+	+	3c	+	+	+
365	57	0.04	0.006	Aspartic acid, 0.5; citric acid, 0.075; glycerol, 1.0	K_2PO_4, 0.5; $MgSO_4 \cdot 7H_2O$, 0.25	3+	3+	3+	4c	0	4c	2c	0	0	0	0
366	57	0.06		Aspartic acid, 0.5; lactic acid, 0.1; glycerol, 1.0	K_2PO_4, 0.5; $MgSO_4 \cdot 7H_2O$, 0.25	3+	3+	3+	2+	3+	0	0	0	0	0	0
367	57	0.09		Aspartic acid, 0.5; tartaric acid, 0.075; glycerol, 1.0	K_2PO_4, 0.5; $MgSO_4 \cdot 7H_2O$, 0.25	3+	3+	3+	2+	+	0	0	0	0	0	0
368	57	0.04	0.008	Aspartic acid, 0.5; malic acid, 0.075; glycerol, 1.0	K_2PO_4, 0.5; $MgSO_4 \cdot 7H_2O$, 0.25	3+	3+	3+	+	0	0	0	0	0	0	0
370	57	0.035	0.003	Aspartic acid, 0.575; glycerol, 1.0	K_2PO_4, 0.5; $MgSO_4 \cdot 7H_2O$, 0.25	3+	3+	3+	3+	2+	0	+	+	+	+	+
371	57	0.053	0.013	Aspartic acid, 0.5; alanine, 0.1; citric acid, 0.1; glycerol, 1.0	K_2PO_4, 0.5; $MgSO_4 \cdot 7H_2O$, 0.25	3+	3+	3+	2+	2+	0	+	4c	0	0	0

372	57	0.025	0.003	Aspartic acid, 0.5; alanine, 0.1; glycerol, 1.5	K_2PO_4, 0.5; $MgSO_4 \cdot 7H_2O$, 0.25	2+	3+	3+	2+	2+	2+	4c	0	0	0	0
373	57	0.15	0.014	Aspartic acid, 0.5; Mg_3 citrate$_2$, 0.25; glycerol, 1.5	$MgSO_4 \cdot 7H_2O$, 0.6; KH_2PO_4, 0.5	2+	3+	3+	3+	2+	+	0	0	0	0	0
374	57	0.13	0.008	Aspartic acid, 0.5; Mg_3 citrate$_2$, 0.25; glycerol, 1.5	$MgSO_4 \cdot 7H_2O$, 0.6; KH_2PO_4, 0.3	3+	2+	2+	0	0	0	4c	0	0	0	0
375	57			Aspartic acid, 0.5; Mg_3 citrate$_2$, 0.25; glycerol, 1.5	$MgSO_4 \cdot 7H_2O$, 0.6; KH_2PO_4, 0.5; K_2CO_3, 1.196	2+	2+	+	+	+	+	2c	0	0	0	0
376	57	0.1		Aspartic acid, 0.1; glycerol, 1.5	$MgSO_4 \cdot 7H_2O$, 0.25; KH_2PO_4, 0.5	3+	3+	4+	4+	3+	3+	+	+	+	+	+
377	57	0.19		Aspartic acid, 0.2; glycerol, 1.5	$MgSO_4 \cdot 7H_2O$, 0.25; KH_2PO_4, 0.5	4+	3+	4+	4+	3+	2+	+	+	+	+	+
378	57	0.115	0.006	Aspartic acid, 0.3; glycerol, 1.5	$MgSO_4 \cdot 7H_2O$, 0.25; KH_2PO_4, 0.5	4+	4+	3+	3+	3+	3+	+	+	+	+	+
379	57	0.12	0.007	Aspartic acid, 0.4; glycerol, 1.5	$MgSO_4 \cdot 7H_2O$, 0.25; KH_2PO_4, 0.5	3+	3+	3+	2+	2+	+	+	5c	3c	0	+
380	57	0.12	0.01	Aspartic acid, 0.5; glycerol, 1.5	$MgSO_4 \cdot 7H_2O$, 0.25; KH_2PO_4, 0.5	3+	3+	3+	0	0	0	2c	0	0	0	0
381	57	0.155	0.01	Aspartic acid, 0.6; glycerol, 1.5	$MgSO_4 \cdot 7H_2O$, 0.25; KH_2PO_4, 0.5	3+	3+	2+	2+	2+	2+	0	0	0	0	0
382	57	0.09		Dextrose, 1.0; citric acid, 0.075; NH_4Cl, 0.2; glycerol, 1.5	KH_2PO_4, 0.5; $MgSO_4 \cdot 7H_2O$, 0.25; K_2SO_4, 0.25; NaCl, 0.12	3+	0	0	0	0	0	0	0	0	0	0
383	57	0.117		Dextrose, 1.0; NH_4Cl, 0.2; glycerol, 1.5	KH_2PO_4, 0.5; $MgSO_4 \cdot 7H_2O$, 0.25; K_2SO_4, 0.25; NaCl, 0.12	3+	0	0	0	0	0	0	0	0	0	0
384	57	0.09	0.002	Dextrose, 1.0; NH_4Cl, 0.2; glycerol, 1.5	$MgSO_4 \cdot 7H_2O$, 0.25; KH_2PO_4, 0.5	3c	0	0	0	0	0	0	0	0	0	0
385	57	0.16		Dextrose, 1.0; $(NH_4)_2SO_4$, 0.2; glycerol, 1.5; Mg_3 citrate$_2$, 0.25	KH_2PO_4, 0.5	2+	+	+	0	0	0	2c	0	0	0	0
386	57	0.114		Dextrose, 1.0; NH_4NO_3, 0.2; glycerol, 1.5	$MgSO_4 \cdot 7H_2O$, 0.25; KH_2PO_4, 0.5	0	0	0	0	0	0	0	0	0	0	0
387	57	0.15		Dextrose, 1.0; $(NH_4)_2$ tartrate, 0.2; glycerol, 1.5	KH_2PO_4, 0.5; $MgSO_4 \cdot 7H_2O$, 0.25; K_2SO_4, 0.25; NaCl, 0.12	0	0	0	0	0	0	0	0	0	0	0
388	57	0.18		Dextrose, 1.0; $(NH_4)_2$ tartrate, 0.2; glycerol, 1.5	$MgSO_4 \cdot 7H_2O$, 0.25; KH_2PO_4, 0.5	0	0	0	0	0	0	0	0	0	0	0
389	57	0.19		Dextrose, 1.0; NH_4 lactate, 0.2; glycerol, 1.5	KH_2PO_4, 0.5; $MgSO_4 \cdot 7H_2O$, 0.25; K_2SO_4, 0.25; NaCl, 0.12	0	0	0	0	0	0	0	0	0	0	0
390[1]	57	0.08	0.005	Mannite, 1.0; NH_4Cl, 0.2; glycerol, 1.5	$MgSO_4 \cdot 7H_2O$, 0.25; KH_2PO_4, 0.5	0	0	0	0	0	0	0	0	0	0	0
391	57	0.16		Mannite, 1.0; $(NH_4)_2$ tartrate, 0.2; glycerol, 1.5	$MgSO_4 \cdot 7H_2O$, 0.25; KH_2PO_4, 0.5	3+	2+	2+	2+	0	0	0	0	0	0	0
392	57	0.14		Mannite, 1.0; $(NH_4)_2$ tartrate, 0.2	$MgSO_4 \cdot 7H_2O$, 0.25; KH_2PO_4, 0.5	3+	3+	3+	2+	0	0	0	+	0	0	0
393	57	0.12		Mannite, 1.0; Mg_3 citrate$_2$, 0.25; glycerol, 1.5; $(NH_4)_2SO_4$, 0.2	KH_2PO_4, 0.5	3+	2+	2+	2+	+	0	5c	0	0	0	0
394[1]	57	0.12		Mannite, 1.0; Mg_3 citrate$_2$, 0.25; $(NH_4)_2SO_4$, 0.2	KH_2PO_4, 0.5	3+	3+	3+	3+	+	0	3c	0	0	0	0
395	57	0.09		Sucrose, 1.0; NH_4Cl, 0.2; glycerol, 1.5	$MgSO_4 \cdot 7H_2O$, 0.25; KH_2PO_4, 0.5	+	+	+	+	0	0	0	0	0	0	0
396	57	0.13		Sucrose, 1.0; $(NH_4)_2$ tartrate, 0.2; glycerol, 1.5	$MgSO_4 \cdot 7H_2O$, 0.25; KH_2PO_4, 0.5	3+	3+	3+	3+	3+	0	+	+	+	0	0
397	57	0.13		Sucrose, 1.0; $(NH_4)_2$ tartrate, 0.2	$MgSO_4 \cdot 7H_2O$, 0.25; KH_2PO_4, 0.5	3+	3+	3+	3+	2+	0	+	4c	2c	+	0
398	57	0.11		Sucrose, 1.0; $(NH_4)_2SO_4$, 0.2; glycerol, 1.5	KH_2PO_4, 0.5	3+	2+	2+	2+	+	+	1c	0	0	0	0
399	57	0.03		Sucrose, 1.0; $(NH_4)_2SO_4$, 0.2	KH_2PO_4, 0.5	2+	2+	0	0	0	0	0	0	0	0	0

[1] The originals of 390–394 contained mannose instead of mannite.

TABLE 21—*Continued*

Proskauer-Beck series—continued

NUMBER OF MEDIUM	BIBLIOGRAPHY	NaOH 2.123M		PERCENTAGE COMPOSITION		RESULTS OF GROWTH										
		Cc. per 5 cc. before sterilization	Cc. per 1 cc. after sterilization	C and N substances	Inorganic substances	First transplant						Second transplant				
						Subcultures:						Subcultures:				
						1	2	3	4	5	6	1	2	3	4	5
400	57	0.09		Lactose, 1.0; NH_4Cl, 0.2; glycerol, 1.5	$MgSO_4 \cdot 7H_2O$, 0.25; KH_2PO_4, 0.5	3+	2+	0	0	0	0	2c	0	0	0	0
401	57	0.15		Lactose, 1.0; $(NH_4)_2$ tartrate, 0.2; glycerol, 1.5	$MgSO_4 \cdot 7H_2O$, 0.25; KH_2PO_4, 0.5	+	0	0	0	0	0	0	0	0	0	0
402	57	0.13		Lactose, 1.0; $(NH_4)_2$ tartrate, 0.2	$MgSO_4 \cdot 7H_2O$, 0.25; KH_2PO_4, 0.5	2+	+	0	0	0	0	0	0	0	0	0
403	57	0.13		Lactose, 1.0; Mg_3 citrate, 0.25; glycerol, 1.5; $(NH_4)_2SO_4$, 0.2	KH_2PO_4, 0.5	3+	2+	+	0	0	0	0	0	0	0	0
404	57	0.17		Lactose, 1.0; Mg_3 citrate, 0.25; $(NH_4)_2SO_4$, 0.2	KH_2PO_4, 0.5	3+	0	0	0	0	0	0	0	0	0	0
405	57	0.12		Raffinose $\cdot 5H_2O$, 1.0; NH_4Cl, 0.2; glycerol, 1.5	$MgSO_4 \cdot 7H_2O$, 0.25; KH_2PO_4, 0.5	+	0	0	0	0	0	0	0	0	0	0
406	57	0.14		Raffinose $\cdot 5H_2O$, 1.0; $(NH_4)_2$ tartrate, 0.2; glycerol, 1.5	$MgSO_4 \cdot 7H_2O$, 0.25; KH_2PO_4, 0.5	2+	2+	2+	2+	2+	2+	3c	3c	0	0	0
407	57	0.17		Raffinose $\cdot 5H_2O$, 1.0; $(NH_4)_2$ tartrate, 0.2	$MgSO_4 \cdot 7H_2O$, 0.25; KH_2PO_4, 0.5	3+	3+	2+	2+	2+	2+	2c	0	0	0	0
408	57	0.12		$(NH_4)_2SO_4$, 0.2; raffinose $\cdot 5H_2O$, 1.0; Mg_3 citrate, 0.25; glycerol, 1.5	KH_2PO_4, 0.5	3+	+	2+	2+	2+	3+	0	0	0	0	0
409	57	0.1		Raffinose $\cdot 5H_2O$, 1.0; Mg_3 citrate, 0.25; $(NH_4)_2SO_4$, 0.2	KH_2PO_4, 0.5	2+	2+	+	2+	2+	2+	2c	0	0	0	0
410	57	0.1		Maltose, 1.0; NH_4Cl, 0.2; glycerol, 1.5	$MgSO_4 \cdot 7H_2O$, 0.25; KH_2PO_4, 0.5	4+	+	0	0	0	0	3c	0	0	0	0
411	57	0.14		Maltose, 1.0; $(NH_4)_2$ tartrate, 0.2; glycerol, 1.5	$MgSO_4 \cdot 7H_2O$, 0.25; KH_2PO_4, 0.5	+	0	0	0	0	0	0	0	0	0	0
412	57	0.13		Maltose, 1.0; $(NH_4)_2$ tartrate, 0.2	$MgSO_4 \cdot 7H_2O$, 0.25; KH_2PO_4, 0.5	4+	2c	0	0	0	0	0	0	0	0	0
413	57	0.15		Maltose, 1.0; Mg_3 citrate, 0.25; glycerol, 1.5; $(NH_4)_2SO_4$, 0.2	KH_2PO_4, 0.5	3+	2+	+	+	+	0	0	0	0	0	0
414	57	0.15		Maltose, 1.0; Mg_3 citrate, 0.25; $(NH_4)_2SO_4$, 0.2	KH_2PO_4, 0.5	3+	2c	0	0	0	0	4c	0	0	0	0
415	57	0.19		$(NH_4)_2$ tartrate, 0.4; glycerol, 1.5	$MgSO_4 \cdot 7H_2O$, 0.25; KH_2PO_4, 0.5	4+	4+	4+	4+	3+	3+	+	0	2c	0	0
416	57	0.21		$(NH_4)_2$ tartrate, 0.4; Mg_3 citrate, 0.25; glycerol, 1.5	$MgSO_4 \cdot 7H_2O$, 0.25; KH_2PO_4, 0.5	3+	3+	3+	3+	3+	3+	3c	0	4c	0	0

417	57	0.1		$(NH_4)_2CO_3 \cdot H_2O$, 0.3; Mg_3 citrate$_2$, 0.25; glycerol, 1.5	$MgSO_4 \cdot 7H_2O$, 0.25; KH_2PO_4, 0.5	3+	4+	4+	4+	3+	2c	+	+	+	+	0
418	57	0.12		$(NH_4)_2SO_4$, 0.3; Mg_3 citrate$_2$, 0.25; glycerol, 1.5	KH_2PO_4, 0.5	2+	2+	+	+	+	0	0	0	0	0	0
419	57	0.13		$(NH_4)_2CO_3 \cdot H_2O$, 0.3; $(NH_4)_2$ tartrate, 0.2; glycerol, 1.5	KH_2PO_4, 0.5; $MgSO_4 \cdot 7H_2O$, 0.25	3+	4+	4+	3+	3+	3+	+	+	+	+	+
420	57	0.25		Citric acid, 0.4; $(NH_4)_2CO_3 \cdot H_2O$, 0.2; glycerol, 1.5	$MgSO_4 \cdot 7H_2O$, 0.25; KH_2PO_4, 0.5	2+	2+	2+	+	0	0	1c	1c	0	0	0
421	57	0.13		NH_4 lactate, 0.5; glycerol, 1.5	$MgSO_4 \cdot 7H_2O$, 0.25; KH_2PO_4, 0.5	2+	2+	2+	2+	2+	2+	0	3c	3c	0	4c
422	57	0.1		Urea, 0.2; glycerol, 1.5	$MgSO_4 \cdot 7H_2O$, 0.25; KH_2PO_4, 0.5	4+	3+	3+	3+	0	3c	+	+	+	0	4c
423	57	0.1		Mannite, 0.6; NH_4Cl, 0.2; glycerol, 1.5	$MgSO_4 \cdot 7H_2O$, 0.25; KH_2PO_4, 0.5	0	0	0	0	0	0	0	0	0	0	0
424	57	0.09	0.006	Mannite, 0.6; NH_4Cl, 0.2	$MgSO_4 \cdot 7H_2O$, 0.25; KH_2PO_4, 0.5	0	0	0	0	0	0	0	0	0	0	0
425	57	0.13		Mannite, 0.6; $(NH_4)_2$ tartrate, 0.2; glycerol, 1.5	$MgSO_4 \cdot 7H_2O$, 0.25; KH_2PO_4, 0.5	3+	3+	3+	3+	2+	2+	5c	3c	0	4c	0
426	57	0.14		Mannite, 0.6; $(NH_4)_2$ tartrate, 0.2	$MgSO_4 \cdot 7H_2O$, 0.25; KH_2PO_4, 0.5	3+	3+	3+	3+	2+	2+	0	0	0	0	0
427	57	0.11		Mannite, 0.6; Mg_3 citrate$_2$, 0.25; $(NH_4)_2SO_4$, 0.2; glycerol, 1.5	KH_2PO_4, 0.5	3+	+	0	0	0	0	0	0	0	0	0
428	57	0.1		Mannite, 0.6; Mg_3 citrate$_2$, 0.25; $(NH_4)_2SO_4$, 0.2	KH_2PO_4, 0.5	+	2c	0	0	0	0	0	0	0	0	0
429	57	0.12		Mannite, 0.6; citric acid, 0.075; NH_4Cl, 0.2; glycerol, 1.5	$MgSO_4 \cdot 7H_2O$, 0.25; KH_2PO_4, 0.5	2+	2+	2+	+	+	+	0	0	0	0	0
430	57	0.11		Mannite, 0.6; citric acid, 0.075; NH_4Cl, 0.2	$MgSO_4 \cdot 7H_2O$, 0.25; KH_2PO_4, 0.5	0	0	0	0	0	0	0	0	0	0	0
17	57	0.02		$(NH_4)_2CO_3 \cdot H_2O$, 0.35; glycerol, 1.5	$MgSO_4 \cdot 7H_2O$, 0.25; KH_2PO_4, 0.15	4+	4+	3+	3+	3+	3+	3c	0	4c	1c	0
352	57	0.05	0.002	Aspartic acid, 0.3; glycerol, 4.0; leucine, 0.4; tyrosine, 0.1	NaCl, 0.5	4c	0	0	0	0	0	0	0	0	0	0
354	57	0.02		NH_4Cl, 0.1; dextrose, 1.0; glycerol, 4.0	NaCl, 0.5	0	0	0	0	0	0	0	0	0	0	0
355	57	0.03		NH_4Cl, 0.1; glucosamine-HCl, 0.2; glycerol, 4.0	NaCl, 0.5	0	0	0	0	0	0	0	0	0	0	0
356	57			Glycerol, 4.0; leucine, 0.4	$MgCl_2$, 0.125; K_2SO_4, trace; $Na_2PO_4 \cdot 12\ H_2O$, 0.25; NaCl, 0.5	1+	2+	1+	1+	+	0	0	+	0	0	0

TABLE 22

Koser-Rettger media

NUMBER OF MEDIUM	BIBLIOGRAPHY	NaOH 2.123M: Cc. per 5 cc. before sterilization	PERCENTAGE COMPOSITION	RESULTS OF GROWTH: First transplant, Subcultures: 1	2	3	4	5	6	Second transplant, Subcultures: 1	2	3	4	5	Third transplant, Subcultures: 1	2	3	4
63	41	0.018	Glycerol, 3.0; $CaCl_2$, 0.01; $MgSO_4 \cdot 7H_2O$, 0.02; K_2HPO_4, 0.1; KH_2PO_4, 0.1; NaCl, 0.5	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
5Bp	41		Medium 63 plus albumen, 0.1	4+	4+	3+	3+	0	0	2+	3+	3+	2+	2+	2c	2c	+	+
105	41	0.035	Medium 63 plus aspartic acid, 0.1	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
106	41	0.017	Medium 63 plus NH_4 lactate, 0.1	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
107	41	0.032	Medium 63 plus glutamic acid, 0.1	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
108	41	0.022	Medium 63 with dextrose, 1.0, instead of glycerol, 3.0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
109	41	0.019	Medium 63 plus tyrosine, 0.1	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
111	41	0.019	Medium 63 plus urea, 0.1	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
112	41	0.023	Medium 63 plus uric acid, 0.1	2+	2+	3+	3+	+	0	0	0	0	0	0	0	0	0	0
113	41	0.025	Medium 63 plus glycerol, 0.1	3+	3+	0	0	0	0	0	0	0	0	0	0	0	0	0
114	41	0.021	Medium 63 plus tryptophane, 0.1	4+	4+	4+	3+	3+	3+	2c	1c	0	0	4c	0	0	0	0
115	41	0.019	Medium 63 plus leucine, 0.1	3+	2+	+	+	0	0	0	0	0	0	0	0	0	0	0
116	41	0.04	Medium 63 plus histidine-HCl, 0.1	4+	4+	4+	4+	4+	4+	+	+	0	0	+	0	0	0	0
3[1]	58	0.009	Albumen, 0.05; $CaCl_2$, 0.1; K_2HPO_4, 0.1; NaCl, 0.5; Na_2SO_4, 0.2	3+	4+	3+	3+	3+	2+	0	2c	0	0	0	0	0	0	0
79[2]	62	0.034	$CaCl_2$, 1.0; KH_2PO_4, 0.1; NaCl, 0.5; Na_2SO_4, 0.2	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
77[3]	6	0.002	$CaCl_2$, trace; K_2PO_4, trace; NaCl, 0.5; Na_2SO_4, 0.1	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
78	6	0.002	Albumen, 0.1; $CaCl_2$, trace; K_2PO_4, trace; NaCl, 0.5; Na_2SO_4, 0.1	4+	4+	3+	3+	3+	3+	3c	0	0	0	0	0	0	0	0
79a	62	0.053	$CaCl_2$, 0.1; KH_2PO_4, 0.1; NaCl, 0.5; Na_2SO_4, 0.2	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0

[1] 3: Cc. of NaOH (2.123 M) per 1 cc. after sterilization = 0.002.

[2] 79: Cc. of NaOH (2.123 M) per 1 cc. after sterilization = 0.002. Sperry and Rettger used only 0.1 per cent $CaCl_2$ (medium 79a).

[3] 77: Bainbridge added 0.1 per cent albumen (see medium 78).

TABLE 23

Sullivan media

NUMBER OF MEDIUM	BIBLIOGRAPHY	NaOH 2.123M: Cc. per 5 cc. before sterilization	PERCENTAGE COMPOSITION	RESULTS OF GROWTH: First transplant: Subcultures: 1	2	3	4	5	6	Second transplant: Subcultures: 1	2	3	4	5
4a	63	0.172	Aspartic acid, 1.0; dextrose, 0.5; $MgSO_4 \cdot 7H_2O$, 0.03; KNO_3, 0.1; K_2HPO_4, 0.1; NaCl, 0.1	4+	2+	2+	0	0	0	0	0	0	0	0
4b[1]	63	0.23	Aspartic acid, 1.5; glycerol, 1.0; $MgSO_4 \cdot 7H_2O$, 0.02; KNO_3, 0.03; K_2HPO_4, 0.1; NaCl, 0.1; NH_4 lactate, 0.08	3+	3+	2+	2+	+	+	2c	2c	0	0	0
89	63	0.164	NH_4 lactate, 0.05; aspartic acid, 1.0; glycerol, 1.0; $MgSO_4 \cdot 7H_2O$, 0.02; KNO_3, 0.02; K_2HPO_4, 0.1; NaCl, 0.5	4+	4+	+	3+	2+	2+	1c	0	0	0	0
171	63	0.156	Aspartic acid, 1.0; glycerol, 2.0; K_2HPO_4, 0.1; Na_2S, 0.2	3+	2+	+	+	+	+	0	0	0	0	0
172	63	0.169	Aspartic acid, 1.0; $MgSO_4 \cdot 7H_2O$, 0.2; K_2HPO_4, 0.1	4+	2+	2+	+	2+	+	0	0	0	0	0
173	63	0.161	Aspartic acid, 1.0; $MgSO_4 \cdot 7H_2O$, 0.02	4+	+	4c	0	0	0	0	0	0	0	0
174	63	0.17	Aspartic acid, 1.0; K_2HPO_4, 0.1; Na_2SO_4, 0.1	0	0	0	0	0	0	0	0	0	0	0
175	63	0.174	Aspartic acid, 1.0; $MgSO_4 \cdot 7H_2O$, 0.02; K_2HPO_4, 0.1	3+	2+	+	5c	0	0	0	0	0	0	0
176	63	0.17	Aspartic acid, 1.0; $MgSO_4 \cdot 7H_2O$, 0.01; K_2HPO_4, 0.1	3+	2+	2+	+	0	0	0	0	0	0	0
177	63	0.175	Aspartic acid, 1.0; $MgSO_4 \cdot 7H_2O$, 0.02; K_2HPO_4, 0.01	3+	+	+	0	0	0	0	0	0	0	0
178	63	0.03	NH_4 lactate, 0.4; $MgSO_4 \cdot 7H_2O$, 0.02; K_2HPO_4, 0.1	0	0	0	0	0	0	0	0	0	0	0
180	63	0.173	Aspartic acid, 1.0; glycerol, 2.0; $MgSO_4 \cdot 7H_2O$, 0.02; K_2HPO_4, 0.1	4+	3+	3+	3+	2+	0	0	0	0	0	0
181	63	0.17	Aspartic acid, 1.0; $MgSO_4 \cdot 7H_2O$, 0.3; NaCl, 1.0	+	0	0	0	0	0	0	0	0	0	0
182	63		Dextrose, 2.0; K_2HPO_4, 0.5; $Na_2PO_4 \cdot 12H_2O$, 0.5	0	0	0	0	0	0	0	0	0	0	0
183	63	0.176	$(NH_4)_2HPO_4$, 1.0; aspartic acid, 1.0; glycerol, 3.5; $MgSO_4 \cdot 7H_2O$, 0.2; K_2HPO_4, 0.1	3+	3+	3+	3+	+	+	0	0	0	0	0
184	63	0.165	Aspartic acid, 1.0; $CaCl_2$, trace; $MgSO_4 \cdot 7H_2O$, 0.02; KNO_3, 0.02; K_2HPO_4, 0.01; NaCl, 0.1	3+	3+	3+	+	+	0	0	4c	0	0	0
185[2]	63	0.04	Aspartic acid, 0.2; mannite, 0.2; $CaCl_2$, trace; $MgSO_4 \cdot 7H_2O$, 0.1; K_2HPO_4, 0.2; NaCl, 0.02	3+	4+	3+	3+	0	0	0	5c	0	0	0
186	63	0.034	Aspartic acid, 0.2; glycerol, 1.0; $MgSO_4 \cdot 7H_2O$, 0.02; K_2HPO_4, 0.1	3+	3+	3+	2+	+	+	0	0	0	0	0
187	63	0.085	Aspartic acid, 0.5; glycerol, 1.0; K_2HPO_4, 0.1	4+	3+	3+	2+	+	+	2c	0	0	0	0
188	63	0.035	Aspartic acid, 0.2; glycerol, 1.0; KNO_3, 0.1; K_2HPO_4, 0.1	2+	2+	2+	0	6c	+	0	0	0	0	0

[1] 4b: Cc. of NaOH (2.123 M) per 1 cc. after sterilization = 0.003.

[2] 185: adaptation from Capaldi and Proskauer (17).

TABLE 23—*Continued*

Sullivan media—Continued

NUMBER OF MEDIUM	BIBLIOGRAPHY	NaOH 2.123M: Cc. per 5 cc. before sterilization	PERCENTAGE COMPOSITION	RESULTS OF GROWTH: First transplant: Subcultures: 1	2	3	4	5	6	Second transplant: Subcultures: 1	2	3	4	5
189p	63	0.049	Aspartic acid, 0.3; glycerol, 2.0; K_2HPO_4, 0.1; NaCl, 0.1	4+	3+	3+	2+	0	0	1c	2c	0	0	0
190	63	0.166	Aspartic acid, 1.0; glycerol, 1.5; KNO_3, 0.05; NaCl, 0.05	4+	3+	3+	3+	3+	3+	1c	0	0	0	0
191	63	0.17	Aspartic acid, 1.0; glycerol, 1.5; KBr, 0.05; K_2HPO_4, 0.1	4+	3+	3+	3+	+	+	0	0	0	0	0
192[a]	63	0.25	Aspartic acid, 2.0; glycerol, 2.0; KI, 0.05; K_2HPO_4, 0.1	4+	+	0	0	0	0	0	0	0	0	0
193	63	0.168	Aspartic acid, 1.0; glycerol, 2.0; KCl, 0.05; K_2HPO_4, 0.1	4+	3+	3+	2+	4c	+	4c	0	0	0	0
194	63	0.166	Aspartic acid, 1.0; glycerol, 2.0; K_2HPO_4, 0.1; K_2SO_4, 0.05	4+	3+	3+	2+	3+	0	4c	0	2c	0	0
195	63	0.164	Aspartic acid, 1.0; glycerol, 2.0; K_2HPO_4, 0.1; Na_2SO_4, 0.05	4+	2+	2+	2+	0	0	1c	0	0	0	0

[a] 192: Cc. of NaOH (2.123 M) per 1 cc. after sterilization = 0.01.

TABLE 24

Uschinsky media

NUMBER OF MEDIUM	BIBLIOGRAPHY	NaOH 2.123M: Cc. per 5 cc. before sterilization	NaOH 2.123M: Cc. per 1 cc. after sterilization	PERCENTAGE COMPOSITION	RESULTS OF GROWTH: First transplant; Subculture: 1	2	3	4	5	6
				Basic mixture: Glycerol, 4.5; $CaCl_2$, 0.01; $MgSO_4 \cdot 7H_2O$, 0.02; K_2HPO_4, 0.1; NaCl, 0.6.						
505	68	0.08		Basic mixture plus NH_4 lactate, 1.0	0	0	0	0	0	0
521	69	0.11		Basic mixture plus $(NH_4)_2$ citrate, 1.0	1c	0	0	0	0	0
522	69	0.25		Basic mixture plus $(NH_4)_2$ tartrate, 1.0	2+	0	0	0	0	0
523	69	0.1	0.014	Basic mixture plus aspartic acid, 1.0	0	0	0	0	0	0
524	69			Basic mixture plus urea, 0.5	0	0	0	0	0	0
525	69	0.02		Basic mixture plus uric acid, 0.025	0	0	0	0	0	0
2	68	0.094	0.01	NH_4 lactate, 1.0; aspartic acid, 0.34; glycerol, 4.0; $CaCl_2$, 0.01; $MgSO_4 \cdot 7H_2O$, 0.02; K_2HPO_4, 0.1; NaCl, 0.6	4+	4+	0	+	+	0[4]
2a	68	0.127		Glycerol, 4.0; NaCl, 0.6; $CaCl_2$, 0.01; $MgSO_4 \cdot 7H_2O$, 0.02; K_2HPO_4, 0.2; NH_4 lactate, 0.6; aspartic acid, 0.34	4+	0	0	0	0	0
57[1]	55	0.07	0.009	Aspartic acid, 0.6; NaCl, 0.5; $Na_2HPO_4 \cdot 12\ H_2O$, 0.2	4+	0	0	0	0	0
58[1]	55	0.089	0.005	NH_4 lactate, 0.6; aspartic acid, 0.4; NaCl, 0.5; $Na_2HPO_4 \cdot 12\ H_2O$, 0.2	2+	0	0	0	0	0
20[2]	61	0.085		NH_4 lactate, 0.5; aspartic acid, 0.25; $CaCl_2$, 0.001; $MgSO_4 \cdot 7H_2O$, 0.001; K_2HPO_4, 0.25; NaCl, 0.25; Na_2SO_4, 0.25	3+	3+	3+	+	2+	+[4]
509	61			$(NH_4)_2HPO_4$, 0.01; Na acetate $\cdot 3H_2O$, 0.5; $MgSO_4 \cdot 7H_2O$, 0.01; K_2HPO_4, 0.2	0	0	0	0	0	0
596	61	0.03		Sucrose, 0.5; $MgSO_4 \cdot 7H_2O$, 0.01; KH_2PO_4, 0.2; NaCl, 0.05	0	0	0	0	0	0
520	72	0.12		NH_4 lactate, 0.6; aspartic acid, 0.4; K_2HPO_4, 0.2; NaCl, 0.8	2c	0	0	0	0	0
75[3]	77	0.051		NH_4 lactate, 0.0063; aspartic acid, 0.34; NaCl, 0.5; $Na_2HPO_4 \cdot 12H_2O$, 0.2	0	0	0	0	0	0

[1] 57 and 58: adaptations from Uschinsky's media, by Park and Williams.

[2] 20: adapted, from Uschinsky's medium, by Smith.

[3] 75: adapted, from Uschinsky's medium, by Wheeler.

[4] Media 2 and 20 showed 2c and + growth, respectively, in the first subculture of the second transplant.

TABLE 25

*Van Delden media**

NUMBER OF MEDIUM	BIBLIOGRAPHY	NaOH 2.123M		PERCENTAGE COMPOSITION	RESULTS OF GROWTH										
					First transplant						Second transplant				
					Subcultures:						Subcultures:				
		Cc. per 5 cc. before sterilization	Cc. per 1 cc. after sterilization		1	2	3	4	5	6	1	2	3	4	5
25	70	0.03		Aspartic acid, 0.1; Na lactate, 0.5; $FeSO_4 \cdot 7H_2O$, trace; $MgSO_4 \cdot 7H_2O$, 0.1; K_2HPO_4, 0.5	3+	2+	3+	2+	2+	3+	0	0	0	0	0
462	70	0.003		Na lactate, 0.1; K_2HPO_4, 0.02	0	0	0	0	0	0	0	0	0	0	0
463	70			Na lactate, 0.05; K_2HPO_4, 0.02	0	0	0	0	0	0	0	0	0	0	0
464	70			K_2HPO_4, 0.02	0	0	0	0	0	0	0	0	0	0	0
465	70	0.006		Ca lactate$_2$, 0.1; K_2HPO_4, 0.05	0	0	0	0	0	0	0	0	0	0	0
466	70	0.023		Aspartic acid, 0.1; Na lactate, 0.5; K_2HPO_4, 0.05	3+	2+	2+	2+	+	1c	0	0	0	0	0
467	70	0.02	0.003	Aspartic acid, 0.1; Na lactate, 0.1; $CaSO_4$, 0.024; K_2HPO_4, 0.05	2+	+	+	0	0	0	0	0	0	0	0
468	70	0.025		Aspartic acid, 0.1; Na lactate, 0.1; $CaSO_4$, 0.048; K_2HPO_4, 0.05	3+	3+	2+	2+	2+	0	+	0	0	0	0
469	70	0.025	0.001	Aspartic acid, 0.1; Na lactate, 0.1; $CaSO_4$, 0.072; K_2HPO_4, 0.05	2+	+	0	0	0	0	0	0	0	0	0
470	70	0.025	0.002	Aspartic acid, 0.1; Na lactate, 0.1; $CaSO_4$, 0.096; K_2HPO_4, 0.05	3+	2+	+	0	0	0	4c	3c	0	0	0
471	70	0.027		$(NH_4)_2SO_4$, 0.02; Na lactate, 0.1; $MgSO_4 \cdot 7H_2O$, 0.06; K_2HPO_4, 0.02	3+	2+	2+	2+	2+	2+	0	0	0	0	0
472	70	0.01		Glycocoll, 0.02; glycerol, 1.5[1]; Na lactate, 0.1; $MgSO_4 \cdot 7H_2O$, 0.06; K_2HPO_4, 0.02	2+	0	0	0	0	0	0	0	0	0	0
474	70	0.015		Dextrose, 0.1; $CaCO_3$, 0.1; $CaSO_4$, 0.096; K_2HPO_4, 0.05	+	0	0	0	0	0	0	0	0	0	0
475	70	0.02		NH_4Cl, 0.05; dextrose, 0.05; $CaSO_4$, 0.096; K_2HPO_4, 0.05	3+	0	0	0	0	0	0	0	0	0	0
476	70	0.02		NH_4Cl, 0.05; Na lactate, 0.05; $CaSO_4$, 0.096; K_2HPO_4, 0.05	3+	0	0	0	0	0	0	0	0	0	0
477	70	0.03		Aspartic acid, 0.5; Na acetate $\cdot 3H_2O$, 0.05; $CaSO_4$, 0.096; K_2HPO_4, 0.05	0	0	0	0	0	0	0	0	0	0	0
478	70	0.015		Aspartic acid, 0.05; Na lactate, 0.05; K_2HPO_4, 0.05	3+	0	0	0	0	0	0	0	0	0	0
482	70	0.025		Aspartic acid, 0.1; Na lactate, 0.25; $MgSO_4 \cdot 7H_2O$, 0.4; K_2HPO_4, 0.05	3+	3+	3+	2+	2+	2+	0	0	0	0	0
483	70	0.014		$(NH_4)_2SO_4$, 0.4[2]; aspartic acid, 0.25; Na lactate, 0.5; K_2HPO_4, 0.05	0	0	0	0	0	0	0	0	0	0	0
486	70	0.02	0.002	Aspartic acid, 0.1; Na$_3$ citrate, 0.25; $CaSO_4$, 0.096; K_2HPO_4, 0.05	2+	0	+	0	0	0	0	0	0	0	0
488	70	0.04		Aspartic acid, 0.1; K_2 tartrate, 0.25; $CaSO_4$, 0.096; K_2HPO_4, 0.05	2+	0	0	0	0	0	0	0	0	0	0
489	70	0.025	0.002	Aspartic acid, 0.1; $CaSO_4$, 0.096; K_2HPO_4, 0.05	2+	3+	3+	+	0	0	0	0	0	0	0
490	70	0.03		Aspartic acid, 0.1; Na lactate, 0.5; $MgSO_4 \cdot 7H_2O$, 0.2; K_2HPO_4, 0.05	2+	2+	2+	2+	2+	2+	+	0	0	0	0

* In all cases where Van Delden used gypsum, $CaSO_4$ in equivalent amounts was substituted.

[1] The original did not use glycerol.

[2] The original contained 0.4 per cent $MgSO_4$, not $(NH_4)_2SO_4$.

TABLE 25—*Continued*

Van Delden—continued

NUMBER OF MEDIUM	BIBLIOGRAPHY	NaOH 2.123M		PERCENTAGE COMPOSITION	RESULTS OF GROWTH										
					First transplant						Second transplant				
					Subcultures:						Subcultures:				
		Cc. per 5 cc. before sterilization	Cc. per 1 cc. after sterilization		1	2	3	4	5	6	1	2	3	4	5
491	70	0.03		Aspartic acid, 0.1; Na lactate, 0.5; $MgSO_4 \cdot 7H_2O$, 0.5; K_2HPO_4, 0.05	3+	3+	3+	3+	0	2c	+	3c	+	0	0
492	70	0.09		Aspartic acid, 0.5; $MgSO_4 \cdot 7H_2O$, 0.2; K_2HPO_4, 0.05	3+	2+	+	+	0	0	0	0	0	0	0
494	70	0.025		Aspartic acid, 0.1; dextrose, 0.2; K_2HPO_4, 0.05	3+	0	0	0	0	0	0	0	0	0	0
495	70	0.03		Aspartic acid, 0.1; Na lactate, 0.5; $MgSO_4 \cdot 7H_2O$, 0.8; K_2HPO_4, 0.05; NaCl, 3.0	0	0	0	0	0	0	0	0	0	0	0
496	70	0.028		Aspartic acid, 0.1; $MgSO_4 \cdot 7H_2O$, 0.8; K_2HPO_4, 0.05; NaCl, 3.0	0	0	0	0	0	0	0	0	0	0	0
497	70	0.03		$(NH_4)_2SO_4$, 0.1; Na lactate, 1.0; $MgSO_4 \cdot 7H_2O$, 0.8; K_2HPO_4, 0.05; NaCl, 3.0	0	0	0	0	0	0	0	0	0	0	0
498	70	0.03	0.015	Aspartic acid, 0.5; $CaSO_4$, 0.96; K_2HPO_4, 0.05	3+	3+	2+	+	+	0	0	0	0	0	0
499	70	0.08	0.003	Aspartic acid, 0.5[3]; Na lactate, 1.0; $MgSO_4 \cdot 7H_2O$, 0.8; K_2HPO_4, 0.05; NaCl, 3.0	0	0	0	0	0	0	0	0	0	0	0
500	70	0.05		Aspartic acid, 0.25; Na lactate, 0.5; $MgSO_4 \cdot 7H_2O$, 0.8; K_2HPO_4, 0.05; NaCl, 3.0	0	0	0	0	0	0	0	0	0	0	0
501	70	0.031		Aspartic acid, 0.1; Na lactate, 0.8; $MgSO_4 \cdot 7H_2O$, 0.8; K_2HPO_4, 0.05; NaCl, 3.0	+	0	0	0	0	0	0	0	0	0	0
502	71	0.03		Aspartic acid, 0.1; Na lactate, 0.5; $MgSO_4 \cdot 7H_2O$, 0.8; K_2HPO_4, 0.05; NaCl, 7.5	0	0	0	0	0	0	0	0	0	0	0
503	71	0.018		NH_4Cl, 0.01[4]; Na lactate, 1.0; K_2HPO_4, 0.05; NaCl, 3.0	0	0	0	0	0	0	0	0	0	0	0
504	71	0.005		NH_4Cl, 0.025; Na lactate, 0.125[5]; $MgSO_4 \cdot 7H_2O$, 0.1; K_2HPO_4, 0.025	0	0	0	0	0	0	0	0	0	0	0

[3] 499: original contained only 0.05 per cent aspartic acid.

[4] 503: original contained only 0.1 per cent NH_4Cl.

[5] 504: original contained 0.25 per cent Na lactate.

TABLE 26

Waksman media

NUMBER OF MEDIUM	BIBLIOGRAPHY	NaOH 2.123M		PERCENTAGE COMPOSITION												RESULTS OF GROWTH										
																First transplant						Second transplant				
																Subcultures:						Subcultures:				
		Cc. per 5 cc. before sterilization	Cc. per 1 cc. after sterilization	NH_4Cl	$(NH_4)_2SO_4$	$CaCl_2$	$Ca_3(PO_4)_2$	$FeSO_4 \cdot 7H_2O$	$MgCl_2$	$MgSO_4 \cdot 7H_2O$	KCl	KH_2PO_4	K_2HPO_4	$NaS_2O_3 \cdot 5H_2O$	S	1	2	3	4	5	6	1	2	3	4	5
50	73	0.046		0.01		0.025			0.01			0.3		0.5		4+	+	0	0	0	0	4c	0	0	0	0
65	74	0.048		0.01		0.025						0.3		0.5		4+	4+	0	0	0	0	4c	0	0	0	0
80	75	0.091			0.2		0.75	Trace		0.05		0.5			1.0	3+	+	0	0	0	0	3c	0	0	0	0
81	73	0.055			0.02	0.025				0.05		0.3			1.0	3+	2+	0	0	0	0	0	0	0	0	0
86	73	0.028			0.02		0.25			0.05		0.1			1.0	4+	3+	2+	2+	0	0	1c	0	0	0	0
150[1]	73			0.01					0.01				0.02	0.5		0	0	0	0	0	0	0	0	0	0	0
151	73	0.063			0.02	0.025		Trace		0.05		0.3			1.0	4+	2+	0	0	0	0	0	0	0	0	0
234	73	0.05		0.01			1.00		0.01			0.3		0.5		4+	4+	4+	3+	6c	0	+	+	4c	2c	0
82	75	0.095			0.2	0.025		0.001		0.05		0.5			1.0	+	0	0	0	0	0	0	0	0	0	0
83	76	0.05	0.002		0.2		0.25	0.001		0.05	0.05		0.1		1.0	+	0	0	0	0	0	0	0	0	0	0
84[2]	76	0.005	0.002		0.2		1.0	0.001		0.05	0.05		0.1		1.0	0	0	0	0	0	0	0	0	0	0	0
85	76	0.009			0.2		1.0	0.001		0.05	0.05		0.1		1.0	3+	+	0	0	0	0	0	0	0	0	0
232	79	0.08			0.2		0.05[3]	0.001		0.05		0.5			1.0	3+	+	+	0	0	0	0	0	0	0	0
646[4]	74	0.212			0.02		0.25	0.001	0.05			1.089			1.0	2+	0	0	0	0	0	0	0	0	0	0

[1] 150: original contained $CaCl_2$, 0.025 per cent; mixture here, like the original, also contained $NaHCO_3$, 0.1 per cent.

[2] 84: also contained dextrose, 0.01 per cent.

[3] 232: original used 0.5 per cent $Ca_3(PO_4)_2$.

[4] 646: original contained M/3 H_3PO_4, 2.0 per cent.

TABLE 27

Nathansohn media

NUMBER OF MEDIUM	BIBLIOGRAPHY	NaOH 2.26M: Cc. per 5 cc. before sterilization	PERCENTAGE COMPOSITION	RESULTS OF GROWTH: First transplant; Subcultures: 1	2	3	4	5	6
			Basic mixture: $MgCl_2$, 0.25; KNO_3, 0.1; NaCl, 3.0; $Na_2HPO_4 \cdot 12H_2O$, 0.05; $Na_2S_2O_3 \cdot 5H_2O$, 0.2						
686	52	0.03	Basic mixture	0	0	0	0	0	0
687	52	0.03	Basic mixture plus sucrose, 0.5	3+	3+	2+	0	0	0
688	52	0.03	Basic mixture plus glycerol, 0.5	0	0	0	0	0	0
689	52	0.03	Basic mixture plus KNa tartrate $\cdot 4H_2O$, 0.2	4+	3+	2+	0	0	0
690	52	0.03	Basic mixture plus K_2 oxalate $\cdot H_2O$, 0.1	0	0	0	0	0	0
691	52	0.025	Basic mixture plus urea, 0.2	0	0	0	0	0	0

TABLE 28

Gibbs media

NUMBER OF MEDIUM	BIBLIOGRAPHY	NaOH 2.26M: Cc. per 5 cc. before sterilization	PERCENTAGE COMPOSITION	RESULTS OF GROWTH: First transplant; Subcultures: 1	2	3	4	5	6
626	32	0.015	$(NH_4)_2SO_4$, 0.1; K_2HPO_4, 0.1; NaCl, 0.2; $MgSO_4 \cdot 7H_2O$, 0.05; $Fe_2(SO_4)_3 \cdot 9H_2O$, trace	0	0	0	0	0	0
627	32	0.005	$Fe_2(SO_4)_3 \cdot 9H_2O$, trace; $MgCO_3$, 0.02; $MgNH_4PO_4 \cdot 6H_2O$, 0.2; $MgSO_4 \cdot 7H_2O$, 0.05; K_2HPO_4, 0.1; NaCl, 0.2	3+	3+	+	+	+	0
628	32	0.005	$Fe_2(SO_4)_3 \cdot 9H_2O$, trace; $MgCO_3$, 0.02; $MgSO_4 \cdot 7H_2O$, 0.05; KCl, 0.2; $NaNH_4HPO_4 \cdot 4H_2O$, 0.34	3+	1c	0	0	0	0
629	32		$Fe_2(SO_4)_3 \cdot 9H_2O$, trace; $MgSO_4 \cdot 7H_2O$, 0.03; K_2HPO_4, 0.05; Na_2CO_3, 0.1; NaCl, 0.05; $NaNO_3$, 0.1	0	0	0	0	0	0

TABLE 29

Long media

NUMBER OF MEDIUM	BIBLIOGRAPHY	NaOH 2.26M: Cc. per 5 cc. before sterilization	PERCENTAGE COMPOSITION	RESULTS OF GROWTH: First transplant, Subcultures: 1	2	3	4	5	6	Second transplant, Subcultures: 1	2	3	4	5
			Basic mixture: $CaCl_2$, trace; glycerol, 5.0; $MgSO_4 \cdot 7H_2O$, trace; KH_2PO_4, 0.2724; NaCl, 0.5											
674	46	0.07	Basic mixture plus $(NH_4)_2$ oxalate $\cdot H_2O$, 1.4208	2c	0	0	0	0	0	0	0	0	0	0
675	46	0.12	Basic mixture plus NH_4 lactate, 1.21 (original used 1.07)	0	0	0	0	0	0	0	0	0	0	0
676	46	0.12	Basic mixture plus NH_4 acetate, 0.7708	0	0	0	0	0	0	0	0	0	0	0
677	46	0.10	Basic mixture plus urea, 0.6004	0	0	0	0	0	0	0	0	0	0	0
678	46	0.05	Basic mixture plus creatinine, 1.132	0	0	0	0	0	0	0	0	0	0	0
679	46	0.10	Basic mixture plus NH_4Cl, 0.5348	0	0	0	0	0	0	0	0	0	0	0
680	46	0.07	Basic mixture plus tyrosine, 1.8112	3+	2+	2+	0	0	0	6c	3c	0	0	0
682	46	0.20	Basic mixture plus histidine-HCl, 1.55	2+	2+	3+	2+	2+	2+	1c	0	0	0	0
683	46	0.10	Basic mixture plus leucine, 1.3116	3+	2+	+	+	+	+	0	0	0	0	0
684	46	0.07	Basic mixture plus alanine, 0.8908	2+	3+	2+	0	0	0	1c	0	0	0	0

TABLE 30

Jordan media

NUMBER OF MEDIUM	BIBLIOGRAPHY	NaOH 2.123M: Cc. per 5 cc. before sterilization	PERCENTAGE COMPOSITION	RESULTS OF GROWTH: First transplant; Subcultures: 1	2	3	4	5	6
27	37	0.04	Aspartic acid, 0.2; $MgSO_4 \cdot 7H_2O$, 0.1; K_2HPO_4, 0.1	3+	3+	2+	0	0	0
649	37	0.05	Aspartic acid, 0.2; NaCl, 0.1	2+	0	0	0	0	0
650	37	0.04	Aspartic acid, 0.2; $CaCl_2$, 0.1	2+	0	0	0	0	0
651	37	0.04	Aspartic acid, 0.2; $MgCl_2$, 0.1	+	0	0	0	0	0
652	37	0.04	Aspartic acid, 0.2; KCl, 0.1	+	0	0	0	0	0
653	37	0.04	Aspartic acid, 0.2; Na_2SO_4, 0.1	2+	0	0	0	0	0
654	37	0.05	Aspartic acid, 0.2; $MgSO_4 \cdot 7H_2O$, 0.1	0	0	0	0	0	0
655	37	0.05	Aspartic acid, 0.2; K_2SO_4, 0.1	0	0	0	0	0	0
656	37	0.04	Aspartic acid, 0.2; NaCl, 0.1; $Na_2PO_4 \cdot 12H_2O$, 0.1	0	0	0	0	0	0
657	37	0.03	Aspartic acid, 0.2; $MgCl_2$, 0.1; $Na_2PO_4 \cdot 12H_2O$, 0.1	4+	3+	3+	0	0	0[1]
658	37	0.045	Aspartic acid, 0.2; $CaSO_4$, 0.1	+	0	0	0	0	0
659	37	0.04	Aspartic acid, 0.2; $Na_2PO_4 \cdot 12H_2O$, 0.1; Na_2SO_4, 0.1	2+	0	0	0	0	0
660	37	0.04	Aspartic acid, 0.2; $MgSO_4 \cdot 7H_2O$, 0.1; $Na_2PO_4 \cdot 12H_2O$, 0.1	3+	0	0	0	0	0
661	37	0.035	Aspartic acid, 0.2; K_2SO_4, 0.1; $Na_2PO_4 \cdot 12H_2O$, 0.1	3+	0	0	0	0	0
662	37	0.03	Aspartic acid, 0.2; $Na_2PO_4 \cdot 12H_2O$, 0.1	3+	0	2c	0	0	0
663	37	0.04	Aspartic acid, 0.2; $Mg_3(PO_4)_2 \cdot 4H_2O$, 0.1	3+	+	2c	0	0	0
664	37	0.04	Aspartic acid, 0.2; K_3PO_4, 0.1	+	0	0	0	0	0
665	37	0.04	Aspartic acid, 0.2; $Ca_3(PO_4)_2$, 0.1	4+	3+	0	0	0	0
666	37	0.05	Aspartic acid, 0.2; $MgSO_4 \cdot 7H_2O$, 0.1; $Na_2PO_4 \cdot 12H_2O$, 0.001	2+	1c	0	0	0	0
667	37	0.03	$MgSO_4 \cdot 7H_2O$, 0.1; $Na_2PO_4 \cdot 12H_2O$, 0.1; NH_4 lactate, 0.5	0	0	0	0	0	0
668	37	0.05	$(NH_4)_3$ citrate, 0.5; $MgSO_4 \cdot 7H_2O$, 0.1; $Na_2PO_4 \cdot 12H_2O$, 0.1	3+	0	0	0	0	0
669	37	0.13	$(NH_4)_2$ tartrate, 0.5; $MgSO_4 \cdot 7H_2O$, 0.1; $Na_2PO_4 \cdot 12H_2O$, 0.1	3+	0	0	0	0	0
670	37		$MgSO_4 \cdot 7H_2O$, 0.1; $Na_2PO_4 \cdot 12H_2O$, 0.1; K_3PO_4, 0.05	0	0	0	0	0	0
671	37		Aspartic acid, 0.2; $Na_2PO_4 \cdot 12H_2O$, 1.0; Na_2SO_4, 0.0001	0	0	0	0	0	0
672	37		Aspartic acid, 0.2; $Na_2PO_4 \cdot 12H_2O$, 0.5; Na_2SO_4, 0.0001	3+	0	0	0	0	0
673	37	0.03	Aspartic acid, 0.2; $Na_2PO_4 \cdot 12H_2O$, 0.2; Na_2SO_4, 0.0001	3+	0	0	0	0	0
685	37	0.03	Aspartic acid, 0.2; $Na_2PO_4 \cdot 12H_2O$, 0.1; Na_2SO_4, 0.0001	3+	+	0	0	0	0

[1] 657: This medium showed 3c and 1c, respectively, in the first and second subcultures of the second transplant.

TABLE 31

Krasnow-Rosenberg media; series I

NUMBER OF MEDIUM	BIBLIOGRAPHY	NaOH 2.123M Cc. per 5 cc. before sterilization	PERCENTAGE COMPOSITION	RESULTS OF GROWTH First transplant Subcultures: 1	2	3	4	5	6
			Basic mixture: $(NH_4)_2CO_3 \cdot H_2O$, 0.5; $MgSO_4 \cdot 7H_2O$, 0.5; K_2HPO_4, 0.5						
15	64	0.06	Basic mixture plus $CaCl_2$, 0.05	2+	2+	2+	0	0	0[1]
15a	O[2]	0.075	Basic mixture plus $CaCl_2$, 0.0025	0	0	0	0	0	0
$15a_2$	O	0.073	Basic mixture plus $CaCl_2$, 0.0005	0	0	0	0	0	0
$15a_3$	O	0.075	Basic mixture	0	0	0	0	0	0
15b	O	0.05	Basic mixture plus NH_4NO_3, 1.5; $CaCl_2$, 0.0025	2+	0	0	0	0	0
$15b_2$	O	0.065	Basic mixture plus NH_4NO_3, 1.5; $CaCl_2$, 0.0005	0	0	0	0	0	0
$15b_3$	O	0.06	Basic mixture plus NH_4NO_3, 1.5	0	0	0	0	0	0
15c	O	0.055	Basic mixture plus $CaCl_2$, 0.0025; KH_2PO_4, 1.0	0	0	0	0	0	0
$15c_2$	O	0.065	Basic mixture plus $CaCl_2$, 0.0005; KH_2PO_4, 1.0	0	0	0	0	0	0
$15c_3$	O	0.045	Basic mixture plus KH_2PO_4, 1.0	0	0	0	0	0	0
15d	O	0.075	Basic mixture plus $CaCl_2$, 0.0025; dextrose, 0.5	0	0	0	0	0	0
$15d_1$	O		Basic mixture plus $CaCl_2$, 0.0005; dextrose, 0.5	0	0	0	0	0	0
$15d_2$	O	0.03	Basic mixture plus dextrose, 0.5	+	0	0	0	0	0
15e	O	0.07	Basic mixture plus $CaCl_2$, 0.0025; KNO_3, 0.1	+	0	0	0	0	0
15f	O	0.06	Basic mixture plus $CaCl_2$, 0.0025; NaCl, 0.1	0	0	0	0	0	0
15g	O		Basic mixture plus NH_4 lactate, 0.5; $CaCl_2$, 0.0025	0	0	0	0	0	0
15h	O	0.03	Basic mixture plus $CaCl_2$, 0.0025; Na_2SO_4, 2.5	0	0	0	0	0	0
15j	O	0.03	Basic mixture plus $CaCl_2$, 0.0025; KCl, 0.05	0	0	0	0	0	0
15k	O	0.03	Basic mixture plus $CaCl_2$, 0.0025; $FeSO_4 \cdot 7H_2O$, 0.001	0	0	0	0	0	0
15l	O	0.035	Basic mixture plus $CaCl_2$, 0.0025; $NaNO_3$, 0.2	0	0	0	0	0	0
15m	O	0.075	Basic mixture plus $CaCl_2$, 0.0025; sucrose, 3.0	2+	1c	0	0	0	0
15o	O	0.07	Basic mixture plus $CaCl_2$, 0.0025; Na lactate, 0.5	2+	+	0	0	0	0
15q	O	0.05	Basic mixture plus $CaCl_2$, 0.0025; M/2 H_2SO_4, 1.0	0	0	0	0	0	0
15r	O	0.01	Basic mixture plus $CaCl_2$, 0.0025; M/3 H_3PO_4, 2.0	0	0	0	0	0	0
15s	O	0.155	Basic mixture plus $CaCl_2$, 0.0025; citric acid, 3.0	2+	2+	2+	2+	2+	3+
15t	O	0.076	Basic mixture plus $CaCl_2$, 0.0025; glycerol, 2.0	0	0	0	0	0	0
15u	O	0.065	Basic mixture plus $CaCl_2$, 0.0002; $Ca_3(PO_4)_2$, 0.0024	2+	2+	+	0	0	0
15v	O	0.057	Basic mixture plus aspartic acid, 0.1; $CaCl_2$, 0.0025	2+	2+	2+	+	0	0[3]
$15a_2a$	O	0.08	$(NH_4)_2CO_3 \cdot H_2O$, 0.5; $CaCl_2$, 0.0005; $MgSO_4 \cdot 7H_2O$, 0.25; K_2HPO_4, 0.5	0	0	0	0	0	0

[1] Medium 15 showed +growth in the first and second subcultures of the second transplant.

[2] "O" in this and subsequent tables indicates that the medium originated with the authors.

[3] Medium 15v showed + growth in the first subculture of the second transplant.

TABLE 32

Krasnow-Rosenberg media; series II

NUMBER OF MEDIUM	BIBLIOGRAPHY	NaOH 2.123M: Cc. per 5 cc. before sterilization	PERCENTAGE COMPOSITION	RESULTS OF GROWTH: First transplant, Subcultures: 1	2	3	4	5	6	Second transplant, Subcultures: 1	2	3	4	5
			Basic mixture: Aspartic acid, 0.2; $MgSO_4 \cdot 7H_2O$, 0.0036; K_2HPO_4, 0.1											
27a	O	0.04	Basic mixture	2+	2+	2+	0	0	0	+	1c	0	0	0
27b[1]	O	0.046	Basic mixture plus NH_4NO_3, 1.5	2+	2+	0	0	0	0	0	0	0	0	0
27c	O	0.095	Basic mixture plus KH_2PO_4, 1.0	2+	2+	2+	0	0	0	1c	0	0	0	0
27c III	O	0.084	Basic mixture plus KH_2PO_4, 1.0; $NaNO_3$, 0.2	4+	0	0	0	0	0	0	0	0	0	0
27c IV	O	0.108	Basic mixture plus KH_2PO_4, 1.0; M/2 H_2SO_4, 1.0	4+	4+	3+	4+	3+	0	0	0	0	0	0
27c V	O	0.115	Basic mixture plus $FeSO_4 \cdot 7H_2O$, 0.001; KH_2PO_4, 1.0	3+	0	0	0	0	0	0	0	0	0	0
27c VI	O	0.083	Basic mixture plus $CaCl_2$, 0.025; KH_2PO_4, 1.0	4+	4+	2+	2+	+	0	+	4c	2c	+	0
27c VII	O	0.081	Basic mixture plus KCl, 0.05; KH_2PO_4, 1.0	4+	4+	3+	2+	+	0	2c	+	0	0	0
27c X	O	0.088	Basic mixture plus KH_2PO_4, 1.0; Na lactate, 0.5	3+	2+	+	+	0	0	0	2+	+	0	0
27c XI	O	0.085	Basic mixture plus $CaCl_2$, 0.005; KH_2PO_4, 1.0	4+	4+	2+	0	0	0	+	+	1c	0	0
27c XIII	O	0.08	Basic mixture plus $Ca_3(PO_4)_2$, 0.001; KH_2PO_4, 1.0	4+	4+	+	0	0	0	6c	3c	0	0	0
27d	O	0.044	Basic mixture plus dextrose, 0.5	2+	2+	2+	0	0	0	+	0	0	0	0
27e	O	0.04	Basic mixture plus KNO_3, 0.1		+	2+	2+	0	0	0	0	0	0	0
27f	O	0.04	Basic mixture plus NaCl, 0.1	2+	+	+	0	0	0	0	0	0	0	0
27g	O	0.063	Basic mixture plus NH_4 lactate, 0.5	2+	2+	2+	0	0	0	+	0	0	0	0
27h	O	0.031	Basic mixture plus Na_2SO_4, 2.5		+	0	0	0	0	0	0	0	0	0
27i	O	0.046	Basic mixture plus $CaCl_2$, 0.5	2+	2+	2+	2+	0	0	+	+	+	+	0
27i I	O	0.04	Basic mixture plus $CaCl_2$, 0.5; KCl, 0.05	4+	4+	2+	2+	+	+	+	0	2c	+	0
27i II	O	0.04	Basic mixture plus $CaCl_2$, 0.5; $FeSO_4 \cdot 7H_2O$, 0.001	4+	4+	4+	4+	4+	+	+	2+	+	+	+
27i III	O	0.04	Basic mixture plus $CaCl_2$, 0.5; $NaNO_3$, 0.2	4+	4+	2+	4+	+	0	+	3c	0	+	0
27i VI	O	0.046	Basic mixture plus $CaCl_2$, 0.5; Na lactate, 0.5	4+	4+	3+	3+	0	0	+	1c	+	0	0
27i VII	O	0.054	Basic mixture plus $CaCl_2$, 0.5; M/2 H_2SO_4, 5.0	4+	4+	3+	3+	2+	0	+	2c	+	1c	+
27i IX	O	0.04	Basic mixture plus $CaCl_2$, 0.05; $Ca_3(PO_4)_2$, 0.001	4+	4+	4+	3+	0	0	+	2+	3+	0	0
27ii	O	0.042	Basic mixture plus $CaCl_2$, 0.01	3+	4+	+	+	0	0	+	0	0	0	0
27ii I	O	0.04	Basic mixture plus $CaCl_2$, 0.01; KCl, 0.05	4+	3+	0	+	0	0	0	0	0	0	0
27ii II	O	0.038	Basic mixture plus $CaCl_2$, 0.01; $FeSO_4 \cdot 7H_2O$, 0.001	2+	+	2+	0	0	0	0	0	0	0	0
27ii III	O	0.037	Basic mixture plus $Ca_3(PO_4)_2$, 0.01; $NaNO_3$, 0.2	+	1c	0	0	0	0	0	0	0	0	0
27ii VI	O	0.035	Basic mixture plus $Ca_3(PO_4)_2$, 0.01; Na lactate, 0.5		+	2+	0	0	0	0	0	0	0	0

[1] Cc. of NaOH (2.123 M) per 1 cc. after sterilization = 0.05.

TABLE 32—*Continued*

Krasnow-Rosenberg media; series II—continued

NUMBER OF MEDIUM	BIBLIOGRAPHY	NaOH 2.123M Cc. per 5 cc. before sterilization	PERCENTAGE COMPOSITION	RESULTS OF GROWTH										
				First transplant						Second transplant				
				Subcultures:						Subcultures:				
				1	2	3	4	5	6	1	2	3	4	5
27ii VII	O	0.066	Basic mixture plus $Ca_3(PO_4)_2$, 0.01; M/2 H_2SO_4, 1.0	+	+	0	0	0	0	0	0	0	0	0
27ii IX	O	0.037	Basic mixture plus $Ca_3(PO_4)_2$, 0.01; citric acid, 0.001	4+	4+	4+	3+	0	0	+	2+	3+	0	0
27j	O	0.039	Basic mixture plus KCl, 0.05	3+	3+	0	0	0	0	0	0	0	0	0
27j I	O	0.038	Basic mixture plus $FeSO_4 \cdot 7H_2O$, 0.001; KCl, 0.05	3+	3+	2+	2+	0	0	0	+	1c	0	0
27j II	O	0.055	Basic mixture plus KCl, 0.05; $NaNO_3$, 0.02	4+	+	1c	0	0	0	0	0	0	0	0
27j V	O	0.039	Basic mixture plus KCl, 0.05; Na lactate, 0.5	2+	3+	3+	2+	0	0	+	3+	3c	0	0
27j VI	O	0.059	Basic mixture plus KCl, 0.05; M/2 H_2SO_4, 1.0	3+	4+	3+	3+	2+	0	+	3+	2+	+	0
27j VIII	O	0.037	Basic mixture plus $Ca_3(PO_4)_2$, 0.001; KCl, 0.0025	4+	4+	2+	0	0	0	0	0	0	0	0
27k	O	0.038	Basic mixture plus $FeSO_4 \cdot 7H_2O$, 0.001	4+	4+	3+	3+	3+	3+	1c	3+	2+	+	0
27k I	O	0.036	Basic mixture plus $FeSO_4 \cdot 7H_2O$, 0.001; $NaNO_3$, 0.2	4+	4+	2+	0	0	0	1c	0	0	0	0
27k IV	O	0.036	Basic mixture plus $FeSO_4 \cdot 7H_2O$, 0.001; Na lactate, 0.5	4+	4+	4+	3+	2+	0	+	+	+	+	0
27k V	O	0.061	Basic mixture plus $FeSO_4 \cdot 7H_2O$, 0.001; M/2 H_2SO_4, 1.0	4+	4+	4+	3+	+	0	0	+	0	3c	0
27k VII	O	0.038	Basic mixture plus $Ca_3(PO_4)_2$, 0.001	4+	4+	2+	4c	+	0	1c	2c	0	0	0
27l	O	0.04	Basic mixture plus $NaNO_3$, 0.2	2+	2+	2+	0	0	0	+	0	0	0	0
27l III	O	0.04	Basic mixture plus Na lactate, 0.5; $NaNO_3$, 0.2	3+	4+	3+	3+	0	0	+	+	2c	0	0
27l IV	O	0.059	Basic mixture plus $NaNO_3$, 0.2; M/2 H_2SO_4, 1.0	3+	4+	2+	2+	0	0	3c	0	0	0	0
27l VI	O	0.035	Basic mixture plus $Ca_3(PO_4)_2$, 0.001; $NaNO_3$, 0.2	4+	4+	3+	+	0	0	2c	0	0	0	0
27m	O	0.04	Basic mixture plus sucrose, 3.0	2+	2+	2+	2+	+	0	+	0	0	0	0
27n	O		Basic mixture plus $(NH_4)_2CO_3 \cdot H_2O$, 0.5; M/H_2SO_4, 0.15	2+	2+	+	0	0	0	+	+	0	0	0
27o	O	0.068	Basic mixture plus Na lactate, 0.5	2+	2+	2+	+	0	0	+	0	0	0	0
27o I	O	0.063	Basic mixture plus Na lactate, 0.5; M/2 H_2SO_4, 1.0	3+	4+	4+	3+	5c	0	1c	0	1c	0	0
27o III	O	0.037	Basic mixture plus $Ca_3(PO_4)_2$, 0.001; Na lactate, 0.5	4+	4+	3+	4+	2+	3+	2c	0	1c	0	3c
27q	O	0.055	Basic mixture plus M/2 H_2SO_4, 1.0	2+	2+	2+	0	0	0	1c	0	0	0	0
27q II	O	0.13	Basic mixture plus $Ca_3(PO_4)_2$, 0.001; M/2 H_2SO_4, 1.0	4+	3+	0	0	0	0	2c	0	0	0	0
27r	O	0.103	Basic mixture plus M/3 H_3PO_4, 2.0	+	+	+	0	0	0	+	0	0	0	0
27s	O	0.253	Basic mixture plus M citric acid, 1.0	2+	2+	2+	+	0	0	0	0	0	0	0
27t	O	0.031	Basic mixture plus glycerol, 2.0	2+	2+	2+	2+	2+	+	+	0	0	0	0
27u	O	0.03	Basic mixture plus $Ca_3(PO_4)_2$, 0.001	2+	2+	2+	0	0	0	+	0	0	0	0

TABLE 33

Miscellaneous series

NUMBER OF MEDIUM	BIBLIOGRAPHY	NaOH 2.123M: Cc. per 5 cc. before sterilization	NaOH 2.123M: Cc. per 1 cc. after sterilization	PERCENTAGE COMPOSITION	RESULTS OF GROWTH: First transplant: Subcultures: 1	2	3	4	5	6	Second transplant: Subcultures: 1	2	3	4	5
145	1	0.11	0.09	NH_4 lactate, 0.6; aspartic acid, 0.8;[1] glycerol, 4.0; NaCl, 0.6; $Na_2PO_4 \cdot 12H_2O$, 0.3	+	+	0	0	0	0	0	0	0	0	0
103	12	0.113	0.002	NH_4 lactate, 1.0; $Na_2HPO_4 \cdot 12H_2O$, 0.2	0	0	0	0	0	0	0	0	0	0	0
104	12	0.003		NH_4 lactate, 1.0; $Na_2HPO_4 \cdot 12H_2O$, 0.2; $CaCO_3$, 1.0	4+	3+	0	0	0	0	0	0	0	0	0
141	13	0.02		Alanine, 0.05; aspartic acid, 0.1; glutamic acid, 0.03; glycocoll, 0.1; tryptophane, 0.15; $CaCl_2$, 0.002; $MgSO_4 \cdot 7H_2O$, 0.05; K_2HPO_4, 0.15	3+	3+	+	+	2c	3c	0	0	0	0	0
437	13	0.003		Tyrosine, 0.075; $CaCl_2$, 0.002; $MgSO_4 \cdot 7H_2O$, 0.02; KNO_3, 0.025; K_2HPO_4, 0.05; K_2SO_4, 0.02	0	0	0	0	0	0	0	0	0	0	0
438	13	0.003		Tryptophane, 0.075; $CaCl_2$, 0.002; $MgSO_4 \cdot 7H_2O$, 0.02; KNO_3, 0.025; K_2HPO_4, 0.05; K_2SO_4, 0.02	0	0	0	0	0	0	0	0	0	0	0
439	14	0.03	0.006	Aspartic acid, 0.3; NaCl, 0.5; $Na_2HPO_4 \cdot 12H_2O$, 0.1; K_2HPO_4, 0.1	+	3c	0	0	0	0	0	0	0	0	0
440	14	0.042	0.01	Aspartic acid, 0.5; dextrose, 3.0;[2] NaCl, 0.5; $Na_2HPO_4 \cdot 12H_2O$, 0.1; K_2HPO_4, 0.1	3+	2+	+	+	0	0	0	1c	0	0	0
62	15	0.036	0.012	$(NH_4)_2CO_3 \cdot H_2O$, 0.1; aspartic acid, 0.45; dextrose, 0.5; glycerol, 2.0; mannite, 0.5; $FeSO_4 \cdot 7H_2O$, 0.003; $MgSO_4 \cdot 7H_2O$, 0.025; $KHSO_4$, 0.025; KH_2PO_4, 0.05; K_2SiO_3, 0.002; $NaNO_3$, 0.1	4+	4+	3+	2+	2+	0	3c	0	2c	0	0
148	29	0.055		Aspartic acid, 0.5; glycerol, 4.0; lactose, 0.3; $MgSO_4 \cdot 7H_2O$, 0.1; K_2HPO_4, 0.1; Na_3 citrate, 0.15	0	0	0	0	0	0	0	0	0	0	0
149	30	0.085		Aspartic acid, 0.5; $MgSO_4 \cdot 7H_2O$, 0.1; K_2HPO_4, 0.1	3+	2+	0	3c	2c	0	0	0	0	0	0
164	36			Histidine-HCl, 0.1; $CaCl_2$, 0.002; $MgSO_4 \cdot 7H_2O$, 0.02; KNO_3, 0.025; K_2HPO_4, 0.5; K_2SO_4, 0.02; $Na_2SO_3 \cdot 7H_2O$, 0.6	3+	3+	2+	0	0	+	4c	3c	0	0	0
71	38	0.175		Aspartic acid, 1.0; dextrose, 1.0; $Na_2HPO_4 \cdot 12H_2O$, 0.2	3c	0	0	0	0	0	0	0	0	0	0
72	38	0.006	0.002	NH_4 lactate, 1.0; dextrose, 1.0; $Na_2HPO_4 \cdot 12H_2O$, 0.2	1c	0	0	0	0	0	0	0	0	0	0
73	38	0.06	0.015	NH_4 lactate, 1.0; dextrose, 1.0; $Na_2HPO_4 \cdot 12H_2O$, 0.1	1c	0	0	0	0	0	0	0	0	0	0

[1] The original used 0.6 per cent aspartic acid.

[2] The original used 0.3 per cent dextrose.

TABLE 33—*Continued*

Miscellaneous series—continued

NUMBER OF MEDIUM	BIBLIOGRAPHY	NaOH 2.123M: Cc. per 5 cc. before sterilization	NaOH 2.123M: Cc. per 1 cc. after sterilization	PERCENTAGE COMPOSITION	RESULTS OF GROWTH: First transplant, Subcultures: 1	2	3	4	5	6	Second transplant, Subcultures: 1	2	3	4	5
170	59	0.034		$(NH_4)_2CO_3 \cdot H_2O$, 0.1; tyrosine, 0.2; glycerol, 2.0; $MgSO_4 \cdot 7H_2O$, 0.1; KH_2PO_4, 0.2; NaCl, 0.5	2+	0	0	0	0	0	0	0	0	0	0
92	60	0.008		$(NH_4)_3$ citrate, 0.1; dextrose, 0.15; $CaCO_3$, 0.1;[1] $FeCl_3 \cdot 6H_2O$, trace; $MgSO_4 \cdot 7H_2O$, 0.02; KH_2PO_4, 0.02; NaCl, 0.02	3+	0	0	0	0	0	0	0	0	0	0
66	65	0.012		Aspartic acid, 0.05; mannite, 0.1; $CaCl_2$, 0.01; $FeCl_3 \cdot 6H_2O$, 0.0002; $MgSO_4 \cdot 7H_2O$, 0.02; KNO_3, 0.05; K_2HPO_4, 0.1; NaCl, 0.01	4+	4+	4+	4+	4+	3+	2+	+	+	+	+
147	66	0.1		$(NH_4)_2SO_4$, 0.2; glycerol, 1.5; mannite, 0.6; $MgSO_4 \cdot 7H_2O$, 0.25; KH_2PO_4, 0.5	2+	0	0	1c	0	0	0	0	0	0	0
297	39			$Ca_3(PO_4)_2$, trace; dextrose, 1.0; $FeSO_4 \cdot 7H_2O$, trace; $MgSO_4 \cdot 7H_2O$, 0.05; KNO_3, 0.24; K_2HPO_4, 0.1; NaCl, 0.02; urea, 0.86	+	0	0	0	0	0	0	0	0	0	0
295	39	0.053		$NH_4H_2PO_4$, 0.142; $Ca_3(PO_4)_2$, trace; dextrose, 1.0; $FeSO_4 \cdot 7H_2O$, trace; $MgSO_4 \cdot 7H_2O$, 0.05; K_2HPO_4, 0.1; NaCl, 0.02	2+	0	0	0	0	0	0	0	0	0	0
625	42	0.032		$Ca(NO_3)_2$, 0.2; glycerol, 3.0; $MgSO_4 \cdot 7H_2O$, 0.05; KCl, 0.05; KH_2PO_4, 0.05	3+	3+	2+	0	0	0	3c	0	0	0	0

[1] The original used excess of $CaCO_3$.

TABLE 34

Miscellaneous media showing no growth

NUMBER OF MEDIUM	BIBLIOGRAPHY	NaOH 2.123M: Cc. per 5 cc. before sterilization	PERCENTAGE COMPOSITION
91	18	0.038	Aspartic acid, 0.1; dextrose, 0.4; KH phthalate, 0.2; $Na_2HPO_4 \cdot 12H_2O$, 0.7
441	19	0.03	NH_4NO_3, 0.5; K_2 tartrate, 0.5
28	25		Medium I: M-CH_3COOH, 3.76 cc., M-NH_4OH, 3.54 cc.; 0.01% $CaCl_2$, 2.0 cc.; 0.01% $FeCl_3 \cdot 6H_2O$, 2.0 cc.; 0.01% $MgSO_4 \cdot 7H_2O$, 2.0 cc.; M-H_3PO_4, 25.9 cc., made up to 100 cc. and 22.4 cc. 2M KOH-NaOH added.
90	35	0.008	$CaCO_3$, 0.1; dextrose, 1.0; $MgSO_4 \cdot 7H_2O$, 0.02; K_2HPO_4, 0.05; NaCl, 0.02.
64	39		Dextrose, 1.0; $FeSO_4 \cdot 7H_2O$, trace; $MgSO_4 \cdot 7H_2O$, 0.05; K_2HPO_4, 0.1; NaCl, 0.2; K_2PO_4, trace; $NaHCO_3$, 1.0.
294	39		Medium 64 plus $Ca_3(PO_4)_2$, trace; KNO_3, 0.029; $NaHCO_3$, 0.641
295	39		Medium 64 plus $Ca_3(PO_4)_2$, trace; KNO_3, 0.243; $NaHCO_3$, 1.323
296	39		Medium 64 plus $Ca_3(PO_4)_2$, trace; urea, 0.86
298	39		Medium 64 plus $Ca_3(PO_4)_2$, trace; $(NH_4)_2SO_4$, 0.19; $NaHCO_3$, 0.17
300	39		Medium 64 plus $Ca_3(PO_4)_2$, trace; NH_4Cl, 0.163; $NaHCO_3$, 0.29
301	39		Medium 64 plus $Ca_3(PO_4)_2$, trace; $(NH_4)_2CO_3 \cdot H_2O$, 0.11
302	39		Medium 64 plus $Ca_3(PO_4)_2$, trace; NH_4 lactate, 0.294; $NaHCO_3$, 0.204
303	39		Medium 64 plus $Ca_3(PO_4)_2$, trace; NH_4 lactate, 0.262; $NaHCO_3$, 0.323
304	39		Medium 64 plus $Ca_3(PO_4)_2$, trace; aspartic acid, 0.19; $NaHCO_3$, 4.506
54	43		$(NH_4)_2SO_4$, 0.5; $MgSO_4 \cdot 7H_2O$, 0.01; K_2PO_4, 0.075
117	49	0.005	$(NH_4)_2SO_4$, 0.1; $FeCl_3 \cdot 6H_2O$, 0.005; $MgSO_4 \cdot 7H_2O$, 0.05; K_2HPO_4, 0.1; NaCl, 0.1
118	49		$FeCl_3 \cdot 6H_2O$, 0.0005; $MgSO_4 \cdot 7H_2O$, 0.05; K_2HPO_4, 0.1; NaCl, 0.1; $NaNO_3$, 0.1
136[1]	67		$MgCl_2$, 0.25; KNO_3, 0.1; NaCl, 3.0; $Na_2HPO_4 \cdot 12H_2O$, 0.05; $Na_2S_2O_3 \cdot 5H_2O$, 0.15
137[1]	67		NH_4Cl, 0.1; $MgCl_2$, 0.01; $NaHCO_3$, 0.1; $Na_2HPO_4 \cdot 12H_2O$, 0.02; $Na_2S_2O_3 \cdot 5H_2O$, 0.5
139	67		$MgCl_2$, 0.01; KNO_3, 0.1; $NaHCO_3$, 0.1; $Na_2HPO_4 \cdot 12H_2O$, 0.02; $Na_2S_2O_3 \cdot 5H_2O$, 0.2
140	67		NH_4Cl, 0.1[2]; $MgCl_2$, 0.01; $NaHCO_3$, 0.1; $Na_2HPO_4 \cdot 12H_2O$, 0.02; $Na_2S_2O_3 \cdot 5H_2O$, 0.2

[1] 136 and 137: adaptations from Nathansohn and Beijerinck, respectively.

[2] The original used 0.01 per cent NH_4Cl.

TABLE 35

Summary of data

ESTIMATE OF GROWTH	FIRST TRANSPLANT						SECOND TRANSPLANT					THIRD TRANSPLANT				FOURTH TRANSPLANT			FIFTH TRANSPLANT		SIXTH TRANSPLANT
	Subcultures:						Subcultures:					Subcultures:				Subcultures:			Subcultures:		Subcultures:
	1	2	3	4	5	6	1	2	3	4	5	1	2	3	4	1	2	3	1	2	1
4+	103	66	29	19	11	6	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
3+	192	103	80	56	33	23	0	4	2	1	0	0	0	0	0	0	0	0	0	0	0
2+	85	98	80	61	52	29	6	6	2	1	1	0	0	0	0	0	0	0	0	0	0
1+	52	45	42	36	41	31	81	34	27	25	21	0	0	1	1	0	0	0	0	0	0
Several colonies	9	16	13	5	8	4	72	44	31	11	8	1	1	0	0	0	0	0	0	0	0
Total no. of media showing growth	441	328	244	177	145	93	159	88	62	38	30	1	1	1	1	0	0	0	0	0	0
Percent of media showing growth	66.5	49.7	39.3	28.6	22.0	14.1	25.6	13.3	9.4	5.7	4.5	0.1	0.1	0.1	0.1	0	0	0	0	0	0

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STUDIES OF THE VALIDITY OF ADVERTISED CLAIMS FOR DENTAL INDUSTRIAL PRODUCTS

II. Lymeogene, A French Tooth Powder[1]

RESEARCH COUNCIL OF THE NEW YORK ACADEMY OF DENTISTRY

WITH THE COLLABORATION OF

CHARLES F. BÖDECKER, D.D.S., F.A.C.D.

New York City

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II. Harmful abrasives in dentifrices..... 173

I

Abstract of the Council's Findings Relating to Lymeogene

FOR THE COUNCIL: WILLIAM J. GIES, CHAIRMAN

1. The damaging effect of abrasives in dentifrices has been brought to the Council's attention.

2. Lymeogene, a French tooth powder that is advertised as being not only devoid of destructive action on enamel but also actually beneficial to it, has been submitted to careful examination. *Fig. 1* presents a reproduction of a portion of one of the trade circulars issued with the aluminum container.[2]

3. Samples of Lymeogene, bought in the open market, contained large proportions of a hard, acicular, crystalline abrasive.

4. This tooth powder cannot be applied to the teeth, as recommended in the trade circulars, without causing severe damage to the enamel.

[1] The Council's first public report was published in the *Journal of Dental Research*, 1927, vii, p. 477.

[2] The name of the manufacturer is not indicated on the packages nor in the accompanying circulars.

PROPRIÉTÉS DU LYMEOGÈNE

La structure délicate des membranes muqueuses aussi bien que péridentales, exige un choix judicieux de l'antiseptique dont on fait usage. En contact avec la salive, LYMEOGÈNE détermine une réaction antiseptique sans effet toxique, augmentant ainsi, au lieu de la diminuer, la résistance des membranes et rendant ce produit particulièrement efficace dans tous les cas d'affections des gencives.

HALITOSIS

En contact avec la salive LYMEOGÈNE dégage de l'oxygène et exerce une action bienfaisante sur les membranes muqueuses de la bouche. LYMEOGÈNE est reconnu comme agissant d'une façon sûre et efficace dans tous les cas d'halitosis. De plus, pour obtenir les meilleurs résultats pour les cures dans les établissements thermaux, il est nécessaire de pratiquer journellement l'hygiène de la bouche avec LYMEOGÈNE avant de prendre les eaux.

PRÉSERVE L'ÉMAIL DES DENTS

LYMEOGÈNE est un puissant prophylactique qui n'attaque pas l'émail des dents, au contraire l'usage fréquent de cette préparation en renforce la structure. Elle possède en outre l'avantage de blanchir les dents graduellement, et de leur rendre leur éclat primitif. LYMEOGÈNE enlève également le film visqueux déposé par la salive sur les dentiers.

SON ACTION SUR LA DENTINE SENSIBLE

La dentine exposée à la salive, surtout dans la bouche des personnes nerveuses, devient hypersensible ce qui empêche de faire un bon prophylactique de la bouche. LYMEOGÈNE, au contact de la salive a une réaction rendant tolérable le travail sur les surfaces sensibles.

SON GOUT

LYMEOGÈNE est d'un goût très spécial qui peut être comparé à celui du pain, c'est-à-dire que l'on ne s'en fatigue jamais. Ce point a été l'objet d'une étude particulière au moment de la composition de cette formule à laquelle furent adjointes des ingrédients minutieusement choisis, grâce à ce procédé, LYMEOGÈNE laisse dans la bouche une sensation délicieuse de fraîcheur et de propreté. Ce dernier point est d'une grande importance pour les fumeurs.

FIG. 1. REPRODUCTION OF ONE PAGE OF A TRADE CIRCULAR IN A PACKAGE OF LYMEOGENE (IN AN ALUMINUM CONTAINER)

Opinions of the individual members of the Council

Concurrent: Henry W. Gillett
Arthur H. Merritt
Bissell B. Palmer, Jr., Secretary
Leuman M. Waugh

Concurrent, with reservations: None

Non-concurrent: None

Unrecorded: Charles F. Bödecker, who succeeded Alfred Owre, resigned, but who conducted the tests before he became a member of the Council.

Holmes C. Jackson (deceased): successor not yet in service.

Acknowledgment

The accompanying paper by Referee Charles F. Bödecker presents some of the scientific data on which these findings are based. The expenses of this study have been met from current appropriations for the support of research in the Dental School of Columbia University. The Council records its appreciation of this important public service, and of Dr. Bödecker's disinterested coöperation.

II

Harmful Abrasives in Dentifrices

CHARLES F. BÖDECKER, D.D.S., F.A.C.D.

Laboratory of Dental Histology and Embryology, Dental School, Columbia University, New York City

Dentifrices are useful primarily to remove harmful and unesthetic deposits from the teeth. For the accomplishment of this purpose by the user, with the least expenditure of energy, manufacturers of dentifrices sometimes incorporate injurious ingredients that readily clean and sometimes even temporarily whiten the teeth. It is only natural that such products should achieve wide popularity. The educated public, however, owing to instructions given by conscientious dentists, are rightfully suspicious of dentifrices that readily whiten teeth.

In dentifrices there are two general types of injurious ingredients that quickly remove deposits and temporarily whiten teeth, namely, (a) acid and (b) grit. Both may be decidedly harmful.

(a) An acid constituent in a dentifrice may readily remove all salivary deposits by softening and dissolving them, but its action may not stop there, for, if active enough—and "strong" mineral acids have been used—it will also attack the enamel. Not only are salivary deposits removed by certain acid tooth-cleaning fluids, but

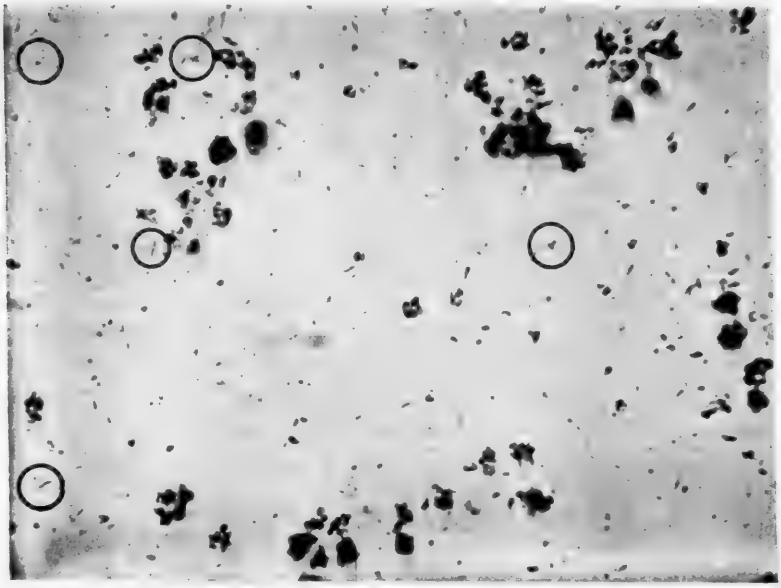

FIG. 2. PHOTOMICROGRAPH OF LYMEOGENE POWDER (TRANSMITTED LIGHT)
Crystals are indicated with circles. *See figs. 3 and 4.*

by some of them the surfaces of teeth are roughened and their predisposition to dental caries is thus increased.[3]

(b) Grit in tooth powders quickly cleans teeth by grinding out surface deposits. That such ingredients are harmful is indicated by the findings below.

Under microscopic examination, Lymeogene,[4] a French tooth

[3] Bödecker: *Journal of Dental Research*, 1927, vii, p. 480.

[4] Bödecker: Proceedings of the International Association for Dental Research, *Ibid.*, 1928, viii, p. 426.

powder, which enjoys the reputation of whitening the teeth of smokers and appears to attract large numbers of American visitors in France, was found to contain minute crystals in the form of stars, spicules, crosses, etc. (*figs. 2* and *3*). These, presumably silicious in nature, are probably obtained either from radiolaria or from a vegetable

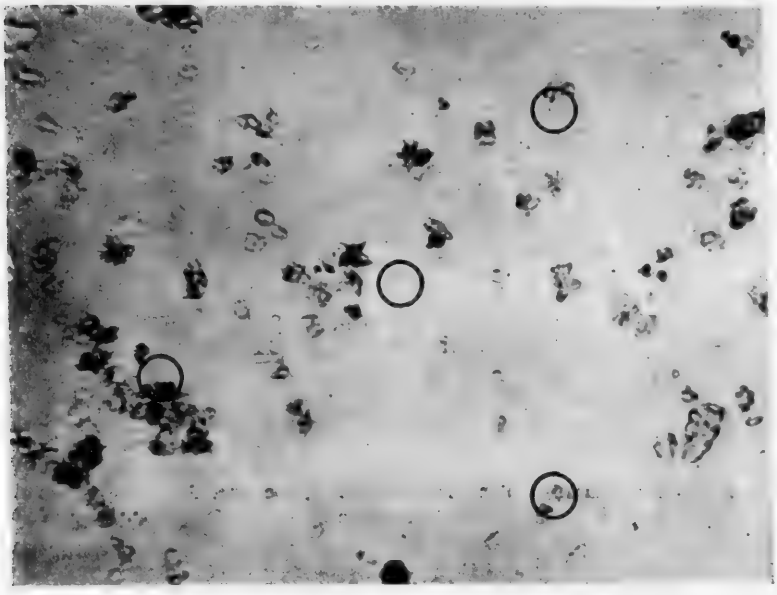

FIG. 3. PHOTOMICROGRAPH OF LYMEOGENE POWDER (POLARIZED LIGHT)
Crystals are indicated with circles. *See figs. 2 and 4.*

silicate. The hardness of silicate is equal to No. 7 of the hardness scale. Dental enamel is softer, equaling No. 6, or apatite, of this scale.

Lymeogene powder was examined under polarized light by Dr. Ed. Applebaum of this Laboratory. The crystals are exceedingly bright under crossed nicols, their form showing even more clearly than by transmitted light (*fig. 3*). In precipitated chalk, photographed (*fig. 4*) under the same conditions as those for the Lymeogene powder shown in *figs. 2* and *3*, there was complete absence of needle-like

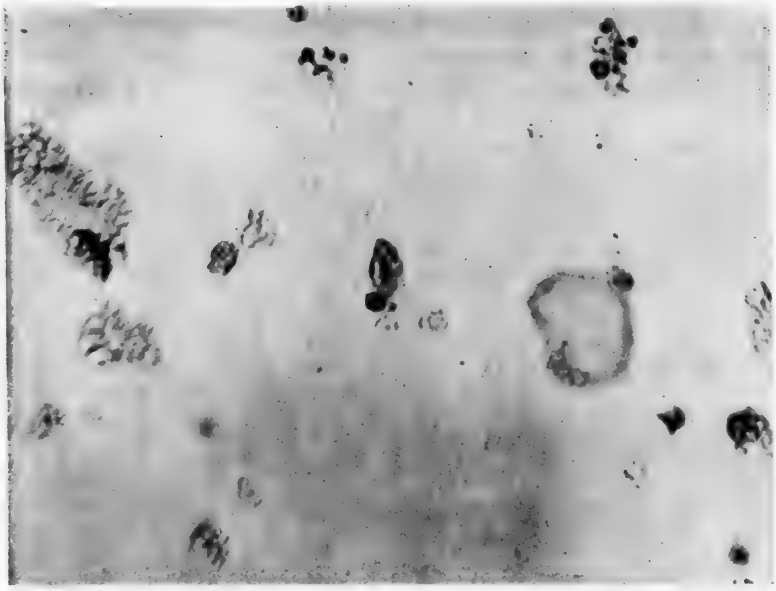

FIG. 4. PHOTOMICROGRAPH OF PRECIPITATED CHALK, UNDER THE SAME CONDITIONS AS THOSE FOR LYMEOGENE IN FIG. 2

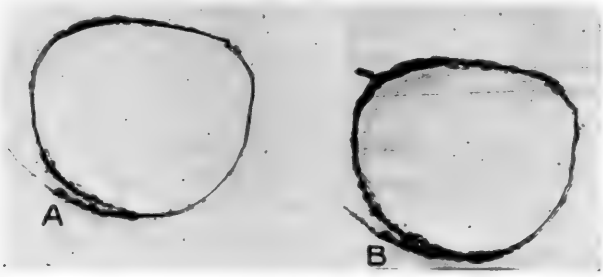

FIG. 5. PHOTOMICROGRAPH OF A SURFACE ON A GLASS SLIDE

A.—Before brushing with Lymeogene. *B*.—After brushing with Lymeogene for ten minutes.

elements. Powdered chalk, being considerably softer than enamel, does not readily abrade the teeth.

If a patient uses a tooth powder that is harder than dental enamel, the teeth must suffer. In order to check this conclusion, Dr. Gies advised me to examine the effect of this tooth powder on glass, which was done in the following manner: upon a clear area on a glass slide a circle was marked with a diamond, as shown in *fig. 5, A*. This area on the slide was then brushed, for a period of ten minutes, with a tooth brush charged with Lymeogene powder and water. After washing and cleaning the slide, examination showed that the hard, sharp-edged crystals in Lymeogene had cut the glass, leaving numerous scratches on its surface, some of which are shown in the accompanying photomicrograph (*fig. 5, B*). The cumulative effect upon enamel of such a powder, as ordinarily used for even a short period, can readily be imagined.

Conclusion. The samples of Lymeogene powder examined in this study contained sharp-edged crystals having a hardness greater than that of dental enamel. A dentifrice containing such an injurious ingredient may seriously damage teeth. Its use should therefore be discouraged.

THE FACE AND TEETH OF MAN[1]

A Study of Growth and Position

MILO HELLMAN, D.D.S., F.A.C.D.

Professor of Orthodontia, New York University, College of Dentistry; Research Associate in Physical Anthropology, American Museum of Natural History, New York City

If expenditure of wealth for a certain thing be taken as an index of the esteem in which it is held, then the human face must be very dear to mankind. More money is spent by people on their faces than on any other bodily feature. According to the latest available statistics reported in newspapers, there are in the nation 40,000 beauty parlors, where the public spends $5,000,000 a day. The investment in the production of machinery, cosmetics, and other supplies used in such establishments is reported to be slightly under $500,000,000. For the purchase of one business, reports the New York Times, an offer of $7,500,000 was "promptly and emphatically rejected." Similar statistics concerning the teeth are not available. Little is known as to just what the public spends for their care. It is, however, difficult to imagine that any living dentist would reject an offer of $7,500,000 for his practice, although it is not unusual to note the attribute of wealth in obituaries of dentists. The regard for teeth, therefore, could not be estimated from the criterion of money spent upon them. But judging from the general clamor of the public for dental service, it is quite obvious that the teeth do come in for a large share of attention. Reports of health examinations in schools, in hospital clinics, in special dental clinics, and even experience in private practice, tend to prove that the teeth are of no less concern to mankind than is the face. A survey of the dental situation made by the New York Tuberculosis and Health Association in Greater New York brought to light some facts of interest in this connection. According to this report there are in

[1] Read at a meeting of the First District Dental Society of the State of New York, New York Academy of Medicine, November 5, 1928.

Greater New York 152 dental clinics, at which service is given free or at a moderate cost. But it is emphasized that this service is entirely inadequate because of insufficient financial support. In one district alone 20,000 children, whose teeth were examined, are waiting to have the necessary dental care.

Here, then, are two situations. One, concerning the face, upon which fortunes are spent; the other, concerning the teeth, which need but do not receive care because of lack of funds. Contrasting as these situations appear, the point to be made is that mankind has become extremely face and tooth conscious. This condition offers an unusual opportunity for the dental profession to demonstrate to the public its interest in mankind, its present position, and its future aims. In the opinion of the public, dentistry is mainly, if not exclusively, concerned with such services only as have for their object the care and repair of diseased teeth; and this for selfish reasons only. It is up to the dental profession to explain that carious teeth, and dental service as rendered today, are only incident to adverse conditions that have arisen in the course of human evolution. Dentistry is groaning under the weight of the burden placed upon it by the task of coping with the present situation. What lightens its present burden is the hope of its aim. The great aim in dentistry is the discovery of the causes of dental decay with the object of preventing it.

The efforts so far exerted in this direction have apparently met with some success. But whether the problem is attacked in the proper way is as yet a matter of conjecture. The propaganda, for example, conducted by the oral hygienist, tends to create the belief that "a clean tooth never decays." This has established an impression that cleanliness and prophylaxis are synonymous. The dental profession is well aware of the fact that there are also other factors of possible etiologic significance in the decay of teeth. The teeth, as is quite well known to the profession, are part of a skeletal system that is susceptible to nutritional influences. It has often been shown that the bones are dependent on proper nutrition for normal development and maintenance. This fact has directed attention to a possible rôle of dietary factors, also, in such manifestations as dental caries.

In an inspection of the school children of Rochester, Minn., reported by L. O. Kappes, rather surprising observations were made. In this

investigation detailed accounts were obtained about maternal conditions during pregnancy, general health factors, past illnesses, environmental influences, and particularly the details of daily diet; i.e., the food habits of the individuals. The investigator reports that the data obtained are not as striking as one would expect. The only feature that seems to be of definite etiologic significance in preventing decay is a diet composed largely of fruits and vegetables. The author reaches the interesting conclusion that heredity, infectious disease, and care of the teeth appear to be of little, if any, significance. (*Amer. Anthro.*, Vol. 91, No. 11, p. 804). It would seem, then, that the propaganda conducted in the attack on the problem of prevention is not based on well founded principles; and the slogan so widely advertised, that "a clean tooth never decays," is as yet lacking the support of reliable scientific proof.

It would seem, then, that a movement of this sort, to be more in keeping with the problem at hand, must rest on more fundamental grounds. Two of the three fundamental manifestations of life, for instance, are nutrition and growth. The face and teeth are particularly susceptible to these factors. An analysis on such grounds is consequently very promising. The face as it is popularly thought of has reference to its outer skin covering only. The attention of the public should be directed to the fact that there is a deeper meaning to it than that. For this purpose a consideration of the facial skeleton is of particular value, because it furnishes the means of showing that the significant features of the face are dependent not so much on its outer covering and what is smeared over it, but rather on its structure. It can, furthermore, be pointed out that the framework of the face embraces not only the facial bones, but also the teeth and their supporting structures. At this point it should be emphasized that the teeth are of importance *not* because they are liable to decay, but rather because they are a part of the framework that constitutes the facial skeleton. In this manner the face and the teeth are brought into a more intimate anatomical relationship. Moreover, since structure and function go hand in hand, it is quite apparent that the closer the structural organization, the more favorable it is for its functional manifestation. The face and teeth may thus be looked upon as an anatomic unit organized for mutual physiologic advantage.

I have often heard the remark: "What do you get out of the study of dead bones, and what have they to do with dentistry?" It is true that skeletal remains present the tissues after death, but it must be remembered that they are the products of life. Moreover, living tissues—even bone—are not to be thought of as static, i.e., fixed and stable. They are systems that are undergoing continual change. Thus, skeletal remains are true records of the stream of life which has come to an end at a particular stage in the course of these changes. The human face, too, undergoes a series of continual changes throughout life. The gradation of these changes, to be sure, is imperceptible, and border lines do not exist; but certain stages, once reached, become well defined and are easily recognized. Thus, anyone will recognize the change of the infant face to that of the boy or the girl, or to that of the adult man or woman. The face in old age and senility is also recognized. Of course, such changes are obvious and apparent to all, but some of the factors concerned in bringing about these changes are not commonly known. For example, few, outside of the dental profession, realize to what extent the teeth are concerned in the modifications of the face. As a matter of fact, the teeth, on account of the space they occupy and the position they assume in the jaw bones, constitute an important factor in these changes. This becomes quite obvious when it is realized that certain changes in the face are coincident with, and perhaps causally related to, certain stages in the development of the teeth. I speak of stages of development, not of age, because they are not necessarily related to age, but rather to certain conditions. Different individuals may reach the same stage of development at different ages. For example, all children in the course of development go through the stage of puberty before they become adults. Some go through it early and others later. This depends on hereditary tendencies, on environmental conditions, and on some other known and many more unknown factors. Likewise, in the child a certain tooth will erupt, but the age at which this takes place will also depend upon similar conditions. The act of eruption of such a tooth will, nevertheless, indicate a definite stage in the development of that dentition. On this basis the development of the face and dentition may be divided into several definite stages as follows:

Stage I. The period of early infancy: designated by the state of development before the deciduous dentition is completed. (*Fig. 1.*)

Stage II. The period of late infancy: designated by the state of development when the deciduous dentition is completed. (*Fig. 2.*)

Stage III. The period of childhood: designated by the state of development when the permanent first molars are erupting or have erupted, in addition to which some or all of the deciduous incisors have been lost and are being replaced by the permanent successors. (*Fig. 3.*)

Stage IV. The period of pubescence: designated by the state of development when the second permanent molars are erupting or have erupted, in addition to which some or all of the deciduous canines and molars have been lost and are being replaced by their permanent successors. (*Fig. 4.*)

Stage V. The period of adulthood: designated by the state of development when the third molars are erupting or have erupted. (*Fig. 5.*)

Stage VI. The period of old age: designated by the state of development when the occlusal surfaces of the molars are worn off to the extent of obliterating the pattern of grooves. (*Fig. 6.*)

Stage VII. The period of senility: designated by the state of development when at least half of the crowns of the teeth are worn off, accompanying or following which some, most, or all of the teeth are lost. (*Fig. 7.*)

It should be noted that stages I to V, inclusive, may be observed in dentitions of all races. Stages VI and VII refer mostly to those races whose food is intermingled with abrasive substances, on account of which the teeth show extensive wear with age. The American Indian and the Australian aboriginal are good examples of these stages because their dentitions are particularly subject to attrition. When such wear is apparent, the rim of the alveolar process becomes thickened. In the civilized white people, the teeth are not so much abraded; but instead of thickening, the margin of the alveolar process undergoes at that stage a good deal of attenuation and atrophy.

If an inquiry be undertaken for the purpose of finding out the nature of the facial changes from stage to stage, and to what extent the teeth participate in those changes, certain measurements are of great help. Of course, the ideal way of making an investigation of this sort would be to measure the same individual as he goes through the different stages. But this is an extremely protracted procedure, and involves a great many difficulties. It is nevertheless being done, and will be reported in due time. A more expedient way is to choose a

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FIG. 1. Skull of American Indian Infant, showing deciduous dentition before its completion. Upper left lateral incisor was lost post mortem.

FIG. 2. Skull of American Indian Infant, showing deciduous dentition after its completion. Upper lateral incisors and canines were lost post-mortem.

FIG. 3. Skull of American Indian Child, showing mixed dentition. The lower deciduous incisors were shed and are replaced by the permanent successors. The permanent first molars have erupted and are in position and occlusion.

FIG. 4. Skull of American Indian Youth, showing the permanent dentition, less the third molars.

FIG. 5. Skull of American Indian Adult, showing complete permanent dentition in normal occlusion. The lower first permanent premolars were lost post-mortem.

FIG. 6. Skull of Old American Indian, showing extensive wear of the crowns of the teeth.

FIG. 7. Skull of Senile American Indian, showing extreme wear of tooth crowns and extensive loss of teeth.

group of skulls favorable for such a study. A collection of skulls of American Indians at the American Museum of Natural History was found to be particularly suitable for this purpose.[2] This collection was chosen (1) because the skulls are rather ancient, dating back at least

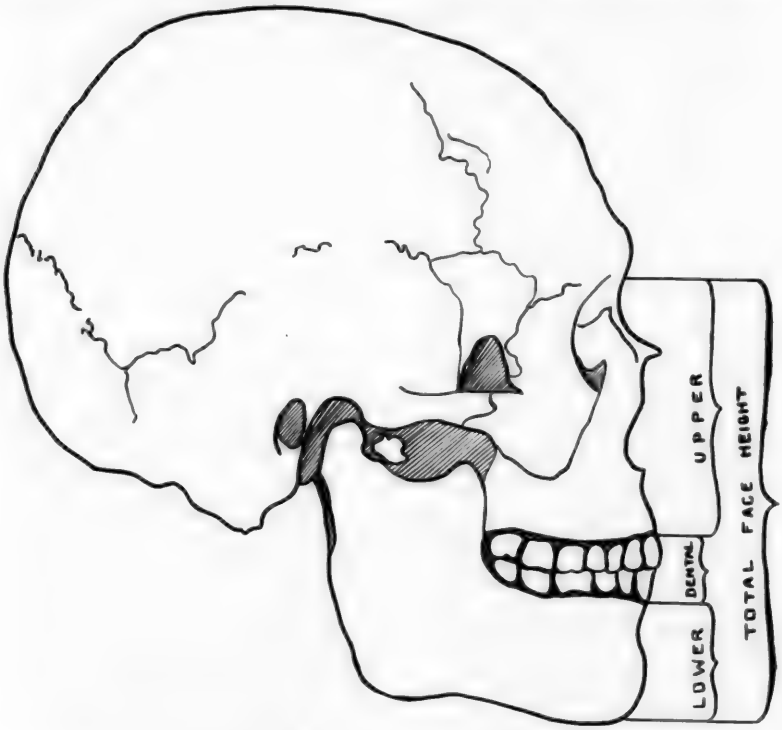

FIG. 8. Outline of skull, showing method of measuring height of face.

2000 years. This is an advantage, because they are unaffected by the artificialities of our modern civilization and so constitute a natural group. (2) They are a homogeneous group; various characters bear evidence of this. (3) They present a complete range of ages from infancy to senility. (4) Their dentitions are far superior and more uniform than the dentitions of modern civilized people. *Figs. 1–9* were made from specimens of this collection.

[2] This collection was excavated in Arizona a few years ago by Earl H. Morris.

Time will not permit a detailed account of this investigation. For the present purpose it will quite suffice to say that the study was pursued on a quantitative basis. In order to gain some understanding of what happens in the course of development, all sorts of measurements had to be taken. Thus height, width, and depth of face, and various angles and positions of the facial mask in relation to the brain case had to be taken into account. It may be mentioned that about 120 measurements of each skull were taken, including measurements of the dentition. As there were 104 specimens, the number of measurements amounted to approximately 12,000.

TABLE 1

Stages of development, showing mean, standard deviation, difference of means, and percentage increase or decrease, in facial height

STAGE	NUMBER	MEAN	STANDARD DEVIATION	ABSOLUTE INCREASE OR DECREASE	PERCENTAGE INCREASE OR DECREASE
		mm.	mm.	mm.	
I	7	66.27	3.42	—	—
II	9	77.80	6.30	11.53	17
III	6	88.35	6.85	10.55	14
IV	6	110.00	6.45	21.65	24
V	13	116.90	9.70	6.90	6
VI	20	121.00	6.70	5.10	4
VII	13	108.10	5.55	−12.90	−11

Some of the dimensions having a direct bearing on the inter-relationship of face and teeth are those pertaining to height. The manner in which these measurements are obtained in shown in *fig. 8*. Thus the total face height is taken from the point where the two nasal bones articulate with the nasal process of the frontal bone (*nasion*) to the lower border of the mandible in the median line (*gnathion*). The upper face height is obtained by measuring from the nasion to the alveolar point between the upper central incisor (*incision*). The lower face height, or symphysial height, is taken from the gnathion to the alveolar point between the lower central incisors. The dental height, or interalveolar area, is measured from the alveolar point of the upper to that of the lower jaw. The measurements thus obtained were grouped according to the various stages of development. Calcu-

lations of averages and standard deviations were made and tabulated to give a clear idea of their meaning. *Table 1* presents the condensed form of the results obtained for the measurement of total face-height.

If the average of measurements of the height of the face (*Table 1*, column 3) is compared from stage to stage, it is found that the dimension increases as we pass from stage I to stage VI. At stage VII there is a decrease. It is, therefore, quite obvious that from early infancy to old age the face keeps on increasing in height; i.e., the face keeps on growing until old age is reached. After this stage there is a decrease in this dimension; i.e., in senility the face goes through a process of

TABLE 2

Stages of development, showing mean and standard deviation of relative proportion of dental height to facial height, expressed in percentage

STAGE	NUMBER	MEAN OF RATIOS	STANDARD DEVIATION
		per cent	*per cent*
I	7	10.71	2.31
II	9	12.89	1.37
III	7	13.71	1.16
IV	6	12.33	1.25
V	13	11.46	1.08
VI	21	10.19	2.34
VII	13	5.92	2.05

degrowth. But growth, it should be observed, is not uniform. For instance, there is a greater increase in late infancy (II) than in childhood (III). In youth the face grows most, after which there is a considerable slowing up during adulthood and still more in old age, after which there is an actual decrease. If these differences in growth are expressed in percentages, then these facts become clearer. Thus, as seen in the last column of *Table 1*, the face increases its height 17 per cent in late infancy, 14 per cent in childhood, 24 per cent in youth, 6 per cent in adulthood and 4 per cent in old age. In senility there is a reversal of this manifestation and the face undergoes a loss of 11 per cent in this dimension.

If, now, the teeth are taken into consideration, it will be found that the changes in the height of the face are not entirely due to the growth of its *bony* framework. The teeth also take part to a large extent in

the increase as well as in the decrease of the dimensions of the face. Thus, as seen in *Table 2*, column 3, the teeth contribute 10.7 per cent of the total face-height in early childhood, but this ratio does not

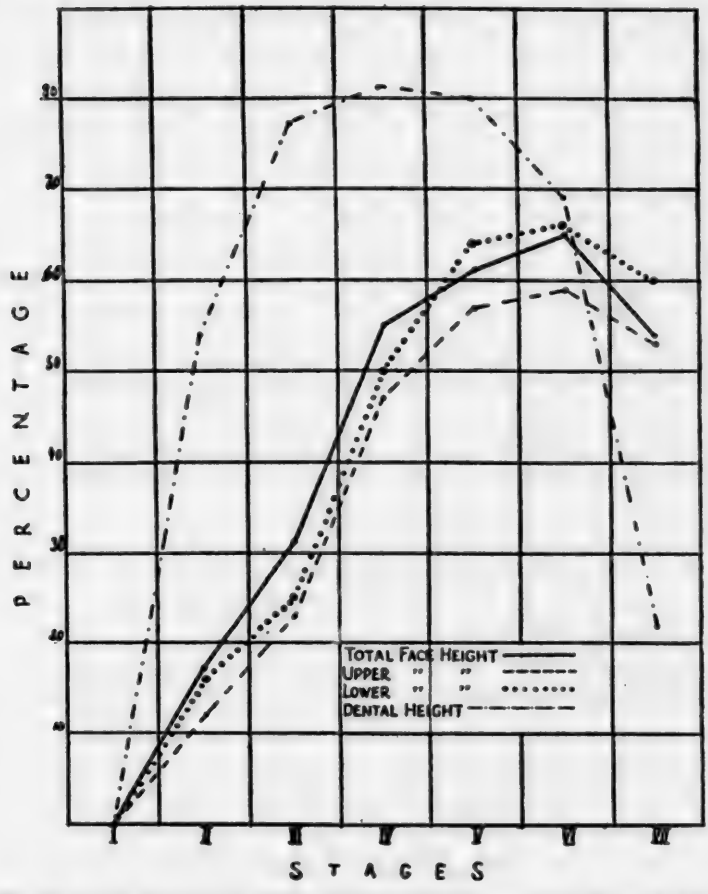

FIG. 9. Growth chart of face height, showing relative increase and decrease in dimension of different parts of the face.

remain the same. The teeth keep on increasing their relative dimension to 13.7 per cent in childhood (III), because they develop at a more rapid rate than does the face. After this stage, the relative dental

height becomes less and less. This gradual decrease from childhood to adulthood is due mainly to the more rapid growth of the face. In old age the decrease is due both to a very slight increase in the dimension of the face and a marked decrease in that of the teeth. In senility, however, there is a decline in the dimensions of both face and teeth, but the teeth decline more than the face. There are then three important facts to be remembered, namely:

(1) The height of the face increases as the individual passes from stage to stage in the course of development until old age is reached, but in senility this dimension is decreased.

(2) The increases are not uniform; i.e., they alternate. Periods of rapid growth are usually followed by periods of slower growth. Thus, the face grows more during late infancy than during childhood, more during youth than during adulthood. Thereafter, growth diminishes very considerably, although it persists until old age.

(3) Different parts grow at different rates; i.e., rapid growth in one part is often accompanied by slower growth of another. Thus, the face increases in height at a different rate than does the dentition. The relationship of dental height to face height thus changes at the different stages.

These facts may be made clearer by the accompanying chart (*fig. 9*), in which the amounts of relative growth of the different parts of the face are plotted. In this chart the horizontal lines indicate the percentage addition or loss of each part; the vertical lines show the stages at which they occur. The parts charted are the total face (solid line), upper face (broken line), lower face (dotted line), and dental height (dot-and-dash line). The chart illustrates the fact that the total face keeps on increasing in height despite the slower growth of either the upper or the lower portions. This is because of the more rapid increase in the dental height during the three earlier stages of development. But despite the early beginning, and slight decline at first in dental height, the face keeps on growing to old age, the lower face growing at a higher rate than the upper. Thereafter the total face dimension declines more rapidly than both the upper and lower portions, because of the excessive decline of the dental height. The face thus increases its height in two ways: (1) by actual growth of its bony framework, and (2) by a sort of wedging apart of the upper from

the lower portions by the more rapid and earlier development of the dentition and its supporting structures. Towards the end, the face diminishes in height by the decline of the bony framework; and also

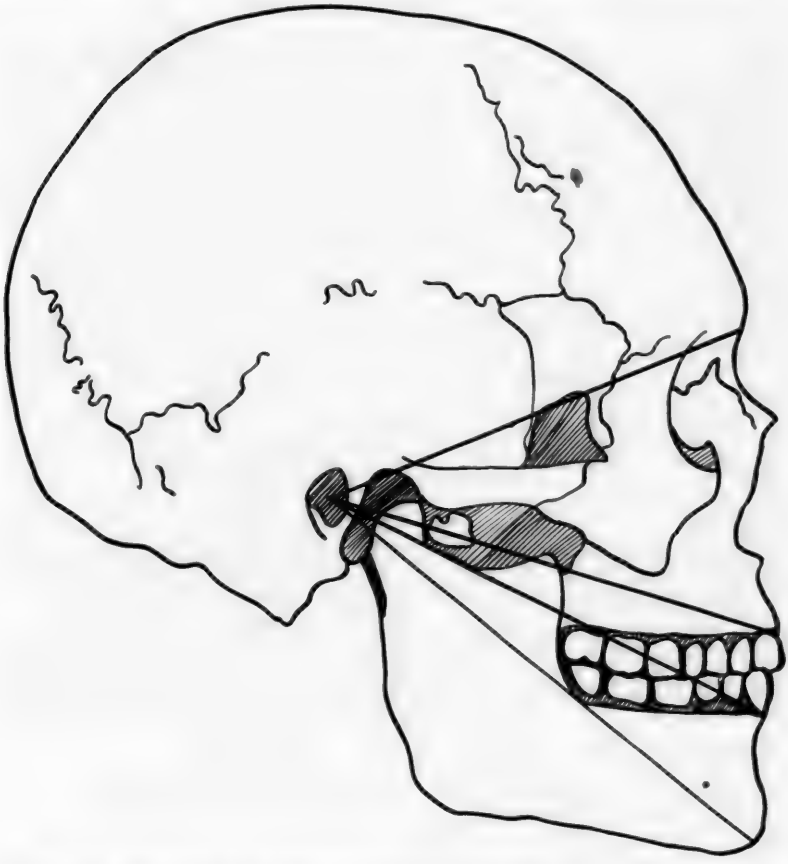

FIG. 10. Outline of skull, showing method of measuring dimension representing position of face.

by the more rapid decline of the dentition, which allows the upper and lower facial parts to come into closer approximation.

These facts should be of interest to certain specialists in dentistry. The orthodontist, for instance, ought to be mindful of the importance

of the periods of rapid growth of the face in late infancy and youth, and of the rapid development of the dentition in childhood. It is during these periods or stages of development that many forms of deficiency, owing to retarded development in earlier stages, may be—

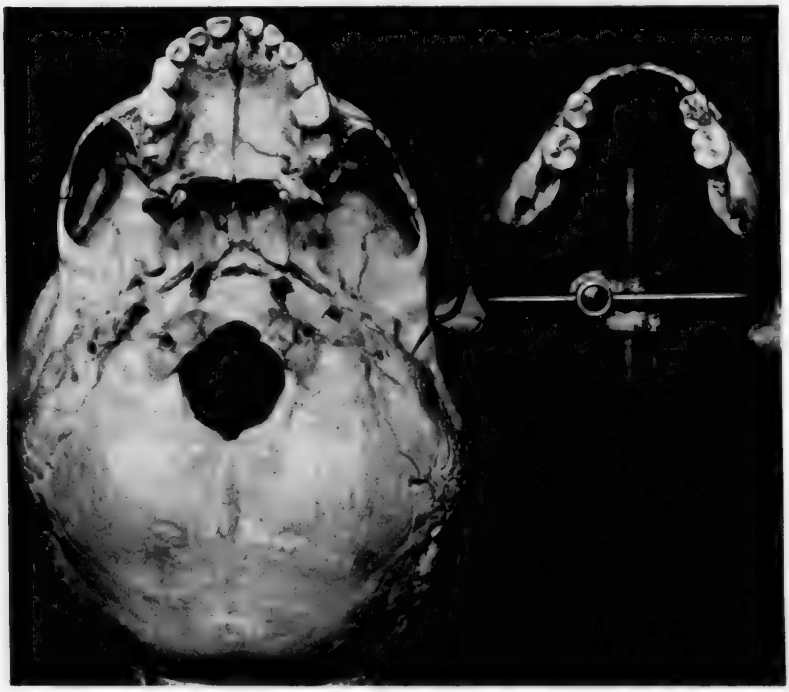

FIG. 11. Skull of White Infant, showing occlusal view of deciduous dentition, posterior to which are seen the areas for the future accommodation of the three permanent molars. In the tuberosities of the upper jaw, the position of the permanent first molar is visible. In the retromolar triangle of the lower jaw, two openings on each side indicate the position for the permanent first and second molars.

and often are—made up by the natural process of more rapid development. The prosthodontist, on the other hand, may be interested to know that results obtained from other studies show that in senility the alveolar process atrophies twice as much in the molar region as in the incisor region, and that the atrophy is approximately one-third greater in the maxilla than in the mandible.

Another aspect of development of interest in this connection is that pertaining to the position of the face in its relation to the brain case, and the position of the teeth in relation to the face. Position as it concerns the face and teeth is ascertainable by means of certain measurements, which may be taken from the external auditory meati on both sides to nasion, prosthion superior, prosthion inferior, and menton, in the sagittal plane, as indicated in *fig. 10*. These measurements, however, are projections of these dimensions and do not represent growth, but rather position. Growth in an antero-posterior direction, it should be remembered, occurs posteriorly; i.e., when the face increases its dimension antero-posteriorly it really grows back-

TABLE 3

Stages of development, showing relative projection of lower three points indicated in fig. 10 beyond the upper point

	RELATIVE PROJECTION BEYOND NASION		
	Pros. sup.	Pros. inf.	Menton
I	−4.2	−3.3	2.1
II	−2.8	−3.5	6.6
III	2.5	8.2	16.2
IV	5.1	12.9	26.0
V	7.9	13.8	28.6
VI	8.3	11.6	26.4
VII	5.0	7.5	24.9

ward. This fact is well illustrated by the growth centers of the jaw bones. These centers are situated in the tuberosity of the maxilla and in the retro-molar triangle of the mandible. *Fig. 11* presents the dental arches of an infant with the deciduous dentition completed (Stage II). Behind the last deciduous molars are seen the areas where the first and second permanent molars are developing. There is as yet insufficient room to accommodate these teeth. But as growth takes place, these areas increase in size. The increase, however, occurs as the portion of the maxilla and mandible anterior to the growth centers move forward; i.e., they depart from the pterygoid processes above and from the ramus below. When this is accomplished, and the first molar takes its position, the growth center

in the same way begins to show the development of the third molar in addition to that of the more advanced state of the second.

As these phenomena of growth appear, the entire facial mask undergoes a change in position. The face really moves and swings, as it were, forward. This forward swing occurs at the highest rate in

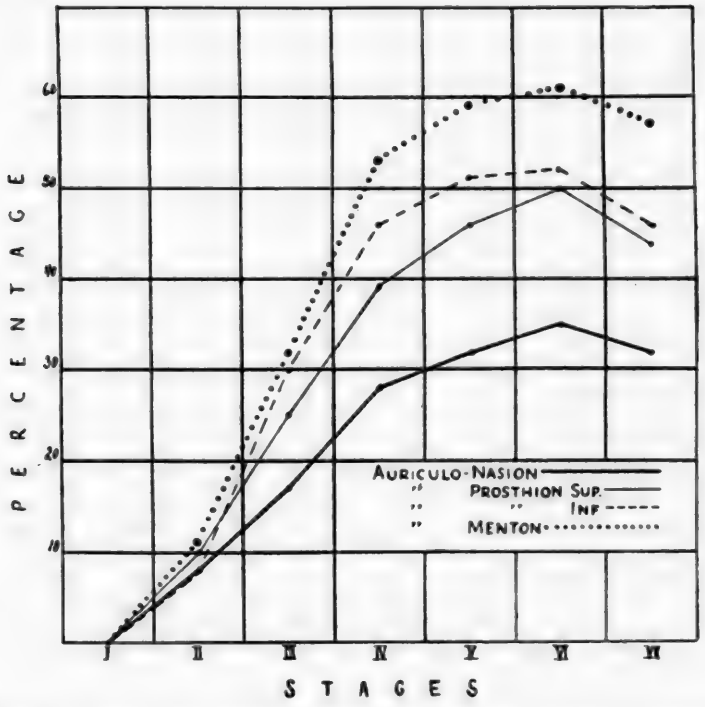

FIG. 12. Growth chart, showing changes of increases and decreases in dimensions indicating relative changes in position of the face.

the lowermost region, and at intermediate rates in the middle regions. Thus, during the course of development, the face and teeth undergo a continual change in their relative position not only to the brain case, but also to each other. The extent of this change may be seen in *Table 3*, giving the relative position of the two dental arches and the mandible as compared to the ear-nasion dimension. Thus, the upper prosthion is at first (Stage I) 4.2 per cent short of this dimension.

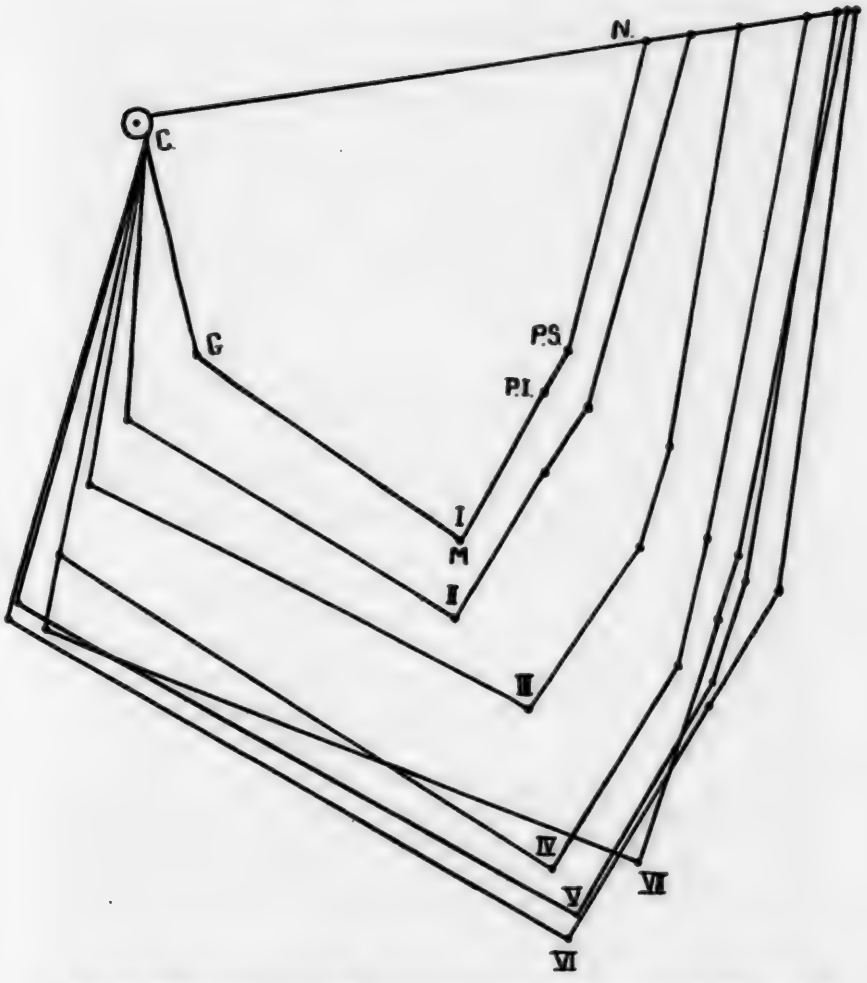

FIG. 13. Diagrammatic profile projection of face, showing changes in position at the different stages of development.

C—Condyle. *G*—Gonion. *M*—Menton. *N*—Nasion. *P.I.*—Prosthion inferior. *P.S.*—Prosthion superior.

But as the face mask moves forward, it diminishes this shortage in Stage II to 2.8 per cent, thereafter increasing its dimension until it surpasses that of the ear-nasion by 8.3 per cent in old age, after which there is a decline. The same thing happens in the lower prosthion. But in this instance the process is more accentuated. The chin region on the other hand, is always beyond that of the ear-nasion distance, and shows during early and middle life the greatest increase and in senility the least decrease. *Fig. 12* illustrates the fact of the forward swing of the face in another way. In this figure the percentage increases and decreases are charted as they occur in each separate dimension. Thus, the ear-nasion dimension increases at the lowest rate, the ear-prosthion superior at a higher rate, the ear-prosthion inferior at a still higher rate, and the ear-menton at the highest rate. By considering these different rates of increase, the manner in which the swing of the facial mask is accomplished becomes quite clear.

Another diagram will illustrate these changes still more clearly. *Fig. 13* represents a diagrammatic profile projection of the face at the various stages of development. This figure was worked out from the means of the measurements of face height, ramus height, antero-posterior dimension of the body of the mandible, and the dimensions of position. It, therefore, combines increases in size with changes in position. This figure quite clearly shows that, as the face goes on developing, the different measured parts increase more in size and also change more in position. But as this is taking place, certain parts increase and change more than others. This creates differences in their dimensions. These differences are due to two factors: time and space. An example of the time factor is manifest during childhood (III), when the face increases its dimension both in size and position more than at any other stage of development. An example of the factor of space is seen in the condition that certain regions increase more than others. Thus, while the face moves forward more in childhood than before or after, the body of the mandible increases in size at that time more than any of the other portions of the face. But while the dimensions keep on increasing in the same approximate direction until old age, there is a notable change in senility. In senility the dentition is either worn down or lost, in consequence of which the size of the face in the total height is reduced, but this is

accompanied by a complete change in position of the mandible. It should, however, be noted that despite the change in position of the mandible, the mandibular angle is not changed materially. It is changed in position only because of the greater approximation of the mandible to the maxilla. The flexion of the mandibular angle, it should be noted, is associated with the relative proportions of the ramus to the body of the mandible. The shorter the former in relation to the latter, the more obtuse the mandibular angle. Thus, as growth progresses and the ramus increases in height relatively more than does the body of the mandible in length, the angle becomes more and more acute. In senility the angle is not greatly modified, it

TABLE 4

Stages of development, showing relationship of ramus height and body length of mandible to mandibular angle

STAGES	AVERAGE RAMUS HEIGHT	AVERAGE MANDIBULAR LENGTH	RELATIVE HEIGHT OF RAMUS TO LENGTH OF MANDIBLE	AVERAGE MANDIBULAR ANGLE
	mm.	*mm.*	*per cent*	*degrees*
I	28.00	51.40	54.47	138.19
II	35.46	58.82	60.29	132.46
III	44.00	79.30	55.49	124.25
IV	53.50	85.44	62.61	122.16
V	60.41	91.92	65.72	117.39
VI	62.40	93.74	66.56	115.65
VII	61.75	91.59	67.42	116.80

only undergoes a marked change in position. *Table 4* clearly illustrates the relationship of ramus height and body length of the mandible to the mandibular angle. It will be noticed that as the mandible grows larger, the ramus increases its proportionate height more and more. Thus, at the beginning (I), the ramus height is only slightly over one-half of the body length of the mandible; at the end (VII), it is more than two-thirds of that length. The only drop in this relative increase occurs during childhood (III), when the growth of the body of the mandible is very much accelerated. Coincident with the relative increase of the ramus height is a decrease in the mandibular angle until old age (VI). In senility, the angle increases again, but only by 1.75°.

The fundamental fact emphasized in *fig. 13* is that, as the face undergoes a change in position, different parts assume different relative

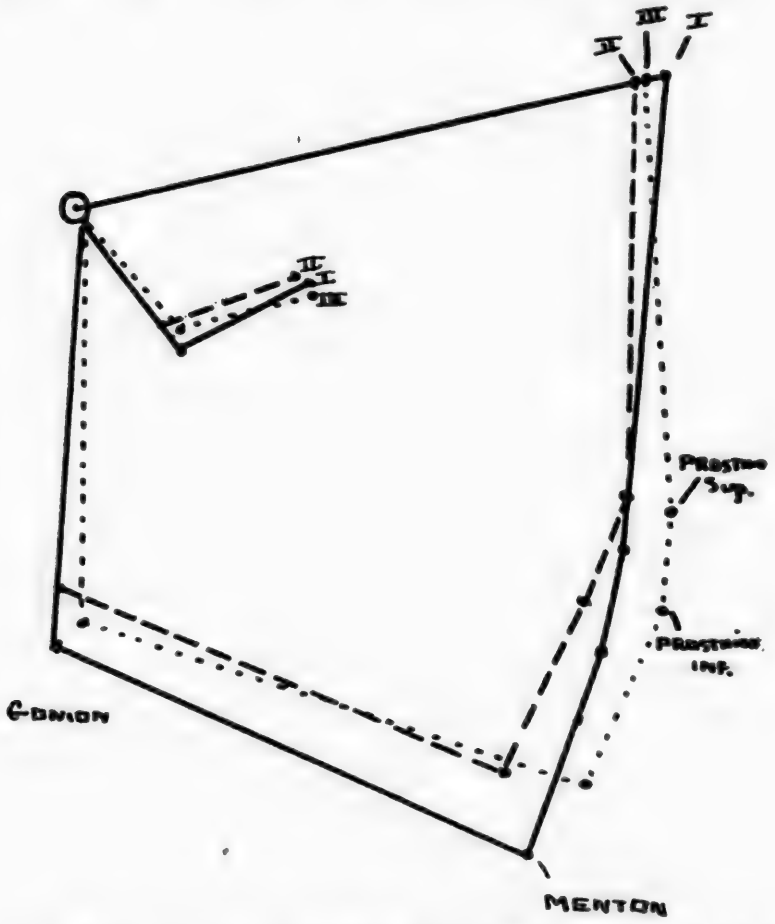

FIG. 14. Diagrammatic profile projection, showing difference in position of face in three racial groups. White, solid line. Australian, dotted line. Hindu, broken line.

positions to it. When the process of development proceeds under favorable conditions, the final result is a normal product, because the

harmonious relation of size and position of one growing part to another is maintained. These relationships may be different in different races. If they are consistent, they bring about differences known as racial

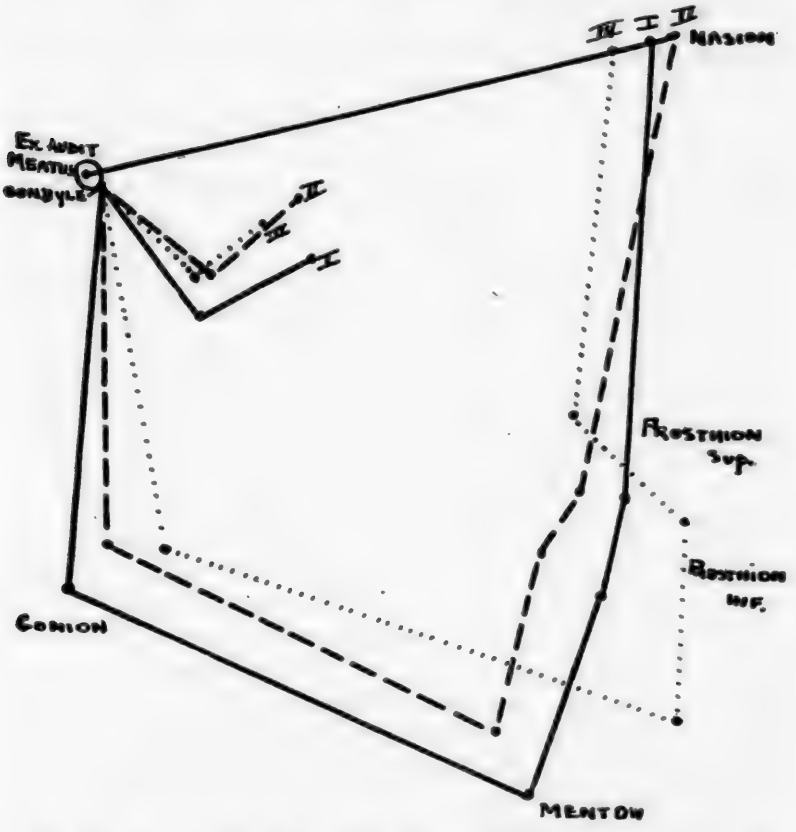

FIG. 15. Diagrammatic profile projection, showing difference in dimension and position of faces of European Whites. *I*, the average of 30 skulls with dentitions in normal occlusion (solid lines). *II*, skull with dentition in Class II, division 1 malocclusion (broken lines). *III*, skull with dentition in Class III malocclusion (dotted lines).

characteristics. A comparison of three contrasting groups, such as Australian Aboriginals, European Whites, and Hindus, illustrates this point very clearly (*fig. 14*). Thus, the face of the Australian (dotted

line) is shorter, the mandible longer, and the dentition projecting farther forward. The face of the European White is longer and more orthognathous. That of the Hindu, although as short as that of the Australian, approximates the White in the upper region, but recedes in the lower. These, however, are normal differences and have reference to types as they are distinguished between one race and another.

Under adverse conditions the growth ratios of interrelated parts are not maintained, and ensuing disturbances modify the features in both size and position. Thus, one part may be retarded in its growth or another accelerated, bringing about not only disproportion, but also malposition. Disproportion and malposition thus become causally related to each other and to the process of growth. Under such conditions, the face as well as the dentition assumes an entirely different aspect. *Fig. 15* illustrates the profile projection of three faces of European Whites, two of which have disturbances of such nature. The profile presented by the solid line is the outline worked out from the mean of 32 skulls with the full complement of teeth in normal occlusion. The face represented by the broken line is that of a skull with the dentition in Class II, division 1 malocclusion. The face represented by the dotted line is that of a skull with the dentition in Class III malocclusion. If the skulls with the dentitions in normal occlusion are taken as the standard and compared with the other two, it may be seen that in the Class II skull the entire face is subnormal in growth and position, while in the Class III skull the upper face is subnormal in both instances, the ramus is relatively small, the body of the mandible is over-developed, and the entire mandible has swung out more anteriorly. It may thus be emphasized that while normal development of face and teeth may bring about differences, they are distinguishable as facial types. Such types may be observed in different races and also among different individuals of the same race. When differences in development become abnormal, they present the same aspect in all racial groups and are distinguishable as malocclusal types.

A summary of the main points in this discussion may be stated as follows:

1. Although the face of the modern human being is given the

greatest attention, it is only from an external aspect, the object being to obscure its real significance by means of superficial camouflage.

2. Although the teeth are now of general concern to mankind, it is just because they are becoming troublesome members in the human economy.

3. Dental caries, and the present services dentistry is able to render, are only incidents brought about by adverse conditions that have arisen in the course of human evolution.

4. The chief aim of the dental profession rests on the endeavor to find out the causes of such conditions, with the object of eliminating them.

5. The real significance of the relationship of face and teeth is not generally recognized.

6. It is the duty of the dental profession to point out the fact that the face and teeth constitute a biologic entity.

7. The relationship between the face and teeth is based on anatomic interdependence for mutual functional advantage.

8. This interdependence is best to be observed in one of those fundamental processes of life which are associated with growth.

9. In the course of development the teeth take a double part in the changes affecting the face:

(a) By the area they occupy, they contribute toward the dimensions of the face;

(b) By the position they assume, they contribute such features as have relation to the type of face.

10. In disturbances of development of the face, the teeth can be used as symptomatic features of considerable diagnostic value.

It may thus be seen that, in pointing out the significance of the face and teeth of man, the practical viewpoint of the dentist has not been considered. The chief object aimed at, nevertheless, was to present an aspect that is of scientific importance to the dental profession.

In conclusion I wish to express my gratitude to the American Museum of Natural History for free access to the wonderful collections harbored there. I am also indebted to the Hamann Museum of the Western Reserve University for the privilege of examining many of the valuable specimens in their collection. Also to my daughter Edith, I am thankful for coöperation in preparing the charts and diagrams used in the illustrations.

57 West 57th Street.

BONE CHANGES AND THEIR SIGNIFICANCE[1]

EGON NEUSTADT, M.U.D. (Vienna)

Department of Orthodontia, New York University, New York City

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I. INTRODUCTION

All tissue changes are produced by cells as a reaction to certain stimuli. The stimuli act either in the direction of higher vitality, causing *productive changes*, which consist of increased production of cells and intercellular substance; or toward diminished vitality, causing *regressive changes*, which are decreased production of cells and intercellular substance, degeneration, and necrosis. In bone the regressive changes are called *resorption*, *halisteresis*, etc.: the productive changes are called *bone condensation*, *condensing osteitis*, *osteosclerosis*, etc. This variety of names has caused much dissatisfaction and confusion, and it indicates that the names cover a multitude of conditions. Furthermore, the current classification into productive and regressive changes is purely descriptive, and without value in diagnosis and treatment.

For scientific as well as practical considerations, bone changes should be classified primarily according to their etiology as *non-pathologic* and *pathologic* changes. The non-pathologic changes are produced by functional adaptations, and therefore do not call for operative interference, while the pathologic changes are brought about by infection and demand surgical treatment. It is, therefore, not so much the phenomena of condensation and rarefaction that interest the dentist

[1] Read at a meeting of the First District Dental Society of the State of New York, New York Academy of Medicine, March 5, 1928; also at a meeting of the International Association for Dental Research, Washington, D. C., March 25, 1928 (*Journal of Dental Research*, 1928, viii, p. 406).

as the question whether the reaction is pathological or not. The answer in one case is a favorable prognosis in periodontic or orthodontic treatment, or the conservative methods of prosthodontia and root-canal therapy; in the other, it is an urgent requirement for surgical treatment.

The differentiation can be made in the roentgenogram and also under the microscope. In the *roentgenogram*, non-pathologic condensation and rarefaction show *gradual transitions* to the sourrounding bone (*figs. 7, 9, 15*). Pathologic condensation and rarefaction, on the other hand, show distinct demarcations from the surrounding bone (*figs. 18, 21, 25*). The *histologic picture* of non-pathologic condensation shows an haversian structure that is more compact than usual, but with canals and systems well outlined (*fig. 1*). Pathologic condensation offers the picture of a bone with disturbed haversian structure. The blood vessels, being the first elements to participate in the struggle against infection, are scarce and irregular; haversian systems cannot be distinguished; the bone lamellae are laid down irregularly, crossing each other in a closely interwoven network (*fig. 2*). The picture resembles the closely interwoven fibres of scar tissue, the parallel being the effort on the part of nature to wall off the infection. Another important feature in the differential diagnosis is the fact that pathologic condensation and rarefaction do not occur independently. At some time in every osseous infection bone structure is destroyed and rarefaction caused by the action of toxins upon cell and tissue elements in their immediate neighborhood. The same toxins, however, induce a different stimulus to cells more remotely situated. While those near the infected center degenerate and die, those farther away respond defensively as long as the toxins are not too strong. The strong bone they build is not for support, but for defense. Every disease offers this duality of phenomena: symptoms that are directly the result of damage, and symptoms that indicate nature's effort to overcome the damage. In local bone infection, condensation may be considered analogous to fever in a general infection.

The process of pathologic change in bone starts, therefore, with *rarefaction*. Then occurs a defensive reaction, and finally there is *rarefaction plus condensation*. The end depends upon the strength of the infection as compared with the tissue resistance. If the infection

FIG. 1. Non-pathologic bone condensation. Haversian canals and systems close together but well outlined. Section made from bone on the tension side of a tooth in malposition; *I, fig. 6.*[2]

[2] All slides were prepared by the ground-section method.

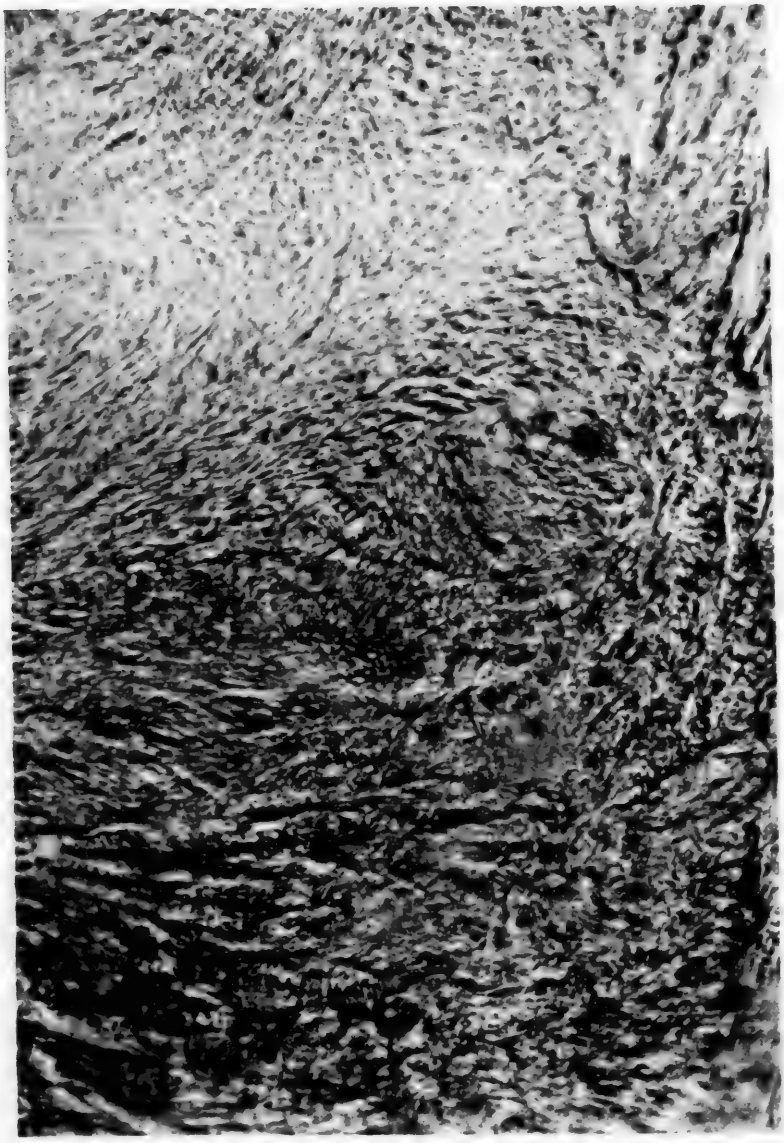

FIG. 2. Pathologic bone condensation (true condensing osteitis); same field and magnification as *fig 1*. No regular arrangement of canals and systems can be seen. The bone layers are arranged irregularly, a few canals are indicated in the lower left part. Section made from bone in periapical area; *III, fig. 22*.

FIG. 3. Reinforcement of the jaw bone under increased function. *L*, functioning side; notice wear on cusp. *R*, side without occlusion. The bone at *L* is heavier in the alveolar process, the cortical plate and the cancellous parts. Section made through molar.

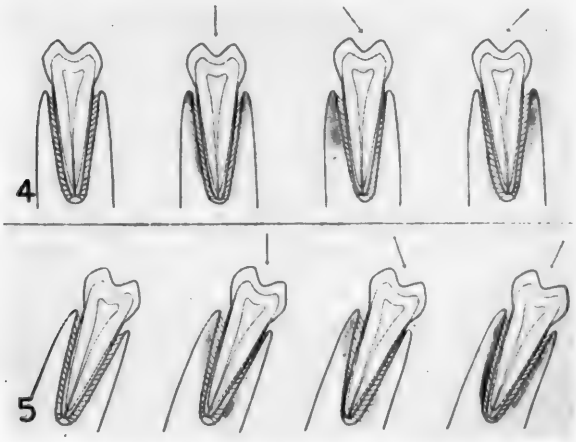

FIG. 4. The tooth is supported by suspension through peridental fibres. The bone functions on the side of tension, indicated through shaded area.

FIG. 5. Tooth in malposition. Teeth in abnormal inclinations obtain their support from localized parts of the alveolar bone, indicated through shaded areas on left side.

is stronger, the rarefaction progresses with a tendency to condensation around the diseased region. If the tissues are stronger, the defensive wall of dense bone progresses toward the center of the infection, and the dead cells and elements are dissolved and carried away.

FIG. 6. Tooth in abnormal inclination. *A*, side of tension; *B*, side of pressure. The bone on the side of tension is well developed and dense; on the side of pressure it is resorbed. High magnification of the dense bone at *I* is shown in *fig. 1*.

If no rarefaction is left, it may be assumed that the process of bone condensation has removed the infection (*figs. 25, 26*).

We shall consider a number of *non-pathologic* changes indicating *gradual transition* of condensation and rarefaction in the roentgenogram, together with photomicrographs of the same specimens showing

FIG. 7. Molar tipped mesially. Strong bone is seen on the distal side (side of tension) near the crest above the fulcrum, and on the mesial side (side of pressure) below the fulcrum. On the side of pressure, there is a rarefaction at the crest.

FIG. 8. Mesio-distal section through molar of *fig. 7;* the extent of the resorption on the mesial side is clearly visible. *M*, mesial; *D*, distal.

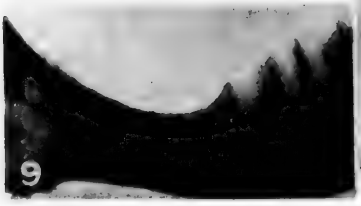

FIG. 9. The jaw bone after loss of posterior teeth. Owing to the change from bending to shearing stresses, the bone is condensed toward the center of the mandible. Section indicated by vertical line is shown in *fig. 10*.

FIG. 10. Dense bone in center of mandible instead of outer border, after loss of molars. *A*, mandibular canal. Section through *I*, *fig. 9*.

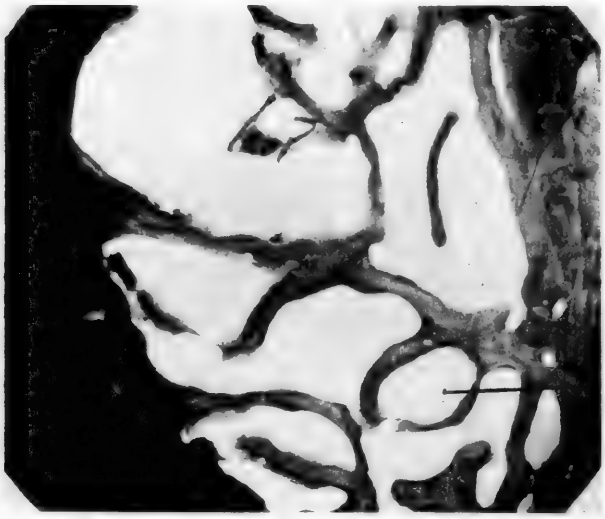

FIG. 11. Normal mandible with dense bone at cortical, cancellous bone at inner, parts. *A*, mandibular canal. Compare with *fig. 10*.

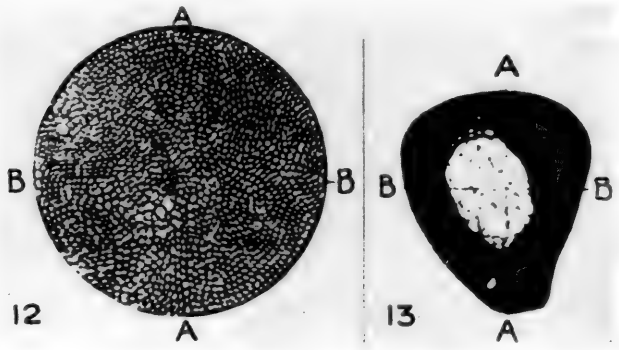

FIG. 12. Section through neck of femur. Owing to shearing movement, bone is distributed in the center. Compare with *fig. 13*. (Koch)

FIG. 13. Section through shaft of femur. Owing to bending stresses, the bone occurs mainly in the outer parts. (Koch)

normal though dense bone. Following this, there will be presented examples of *pathologic changes* that include rarefaction and condensation combined, *clearly marked* in the roentgenogram, and easily discernible as *infected bone* in the slide. There is also included one example of an infection that subsided and left a condensation with distinct outline and pathologic bone.

II. NON-PATHOLOGIC CHANGES

1. Reinforcement of the jaw bone under *increased function* (*fig. 3*). Under the influence of unilateral mastication the bony structure of the

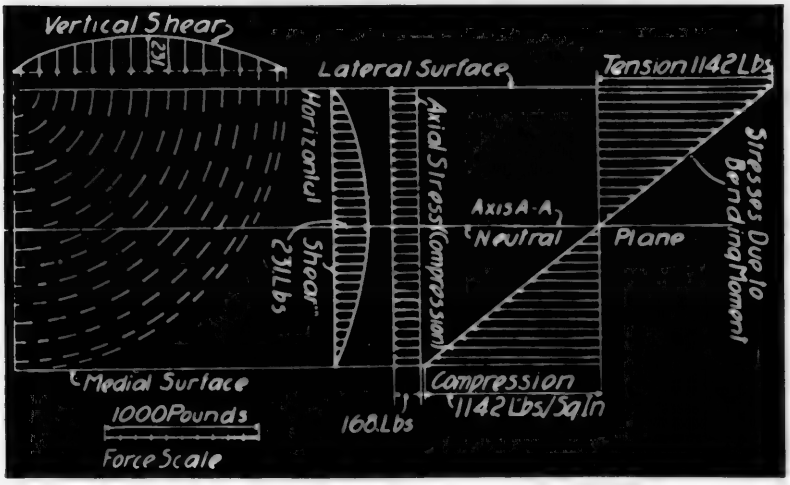

FIG. 14. Diagram of stresses. Shearing stresses reach their maximum in the neutral axis, bending stresses attain the maximum farthest away from the neutral axis. (Koch)

functioning side has been strengthened. The bone is heavier not only adjacent to the tooth, but at points distant from it, in the cancellous parts of the center, and in the cortical parts.

2. Localized condensation of the alveolar process of *teeth that are in malposition*, or receive lateral traumatic stress (*figs. 4, 5*). The support of the tooth is furnished chiefly through suspension by peridental fibres. The bone, therefore, is functioning on the side of tension, while being resorbed on the other through pressure (*fig. 6*). Below the

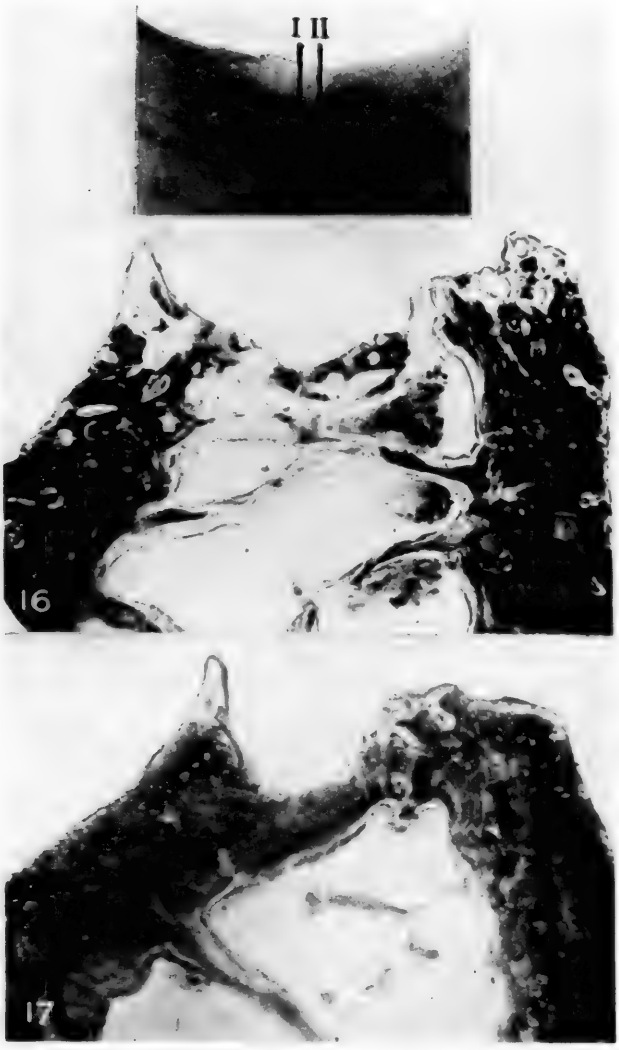

FIG. 15. Rarefied area seen after extraction of teeth. Section through *I* is shown in *fig. 16;* section through *II* is shown in *fig. 17*.

FIG. 16. One stage in the process of non-pathologic bone changes after extraction of teeth. The socket fills in slowly from the center while the crest is gradually absorbed. Section through *I*, *fig. 15*.

FIG. 17. Another phase of the elimination of the alveolar process. Through the divergence in productive changes from the socket and regressive changes from the crest, a space appears in the center of the upper border that simulates a rarefaction. Section through *II*, *fig. 15*.

fulcrum of the tooth, the sides of tension and pressure change, and there is a corresponding alteration in the structure of the alveolar bone. Molars that have lost their mesial contact, and have tipped forward, show the same phenomenon (*figs. 7, 8*). We also notice here a modification in the jaw bone, where teeth have been lost, owing to a change in function, which is still better exemplified in the next case (*fig. 9*).

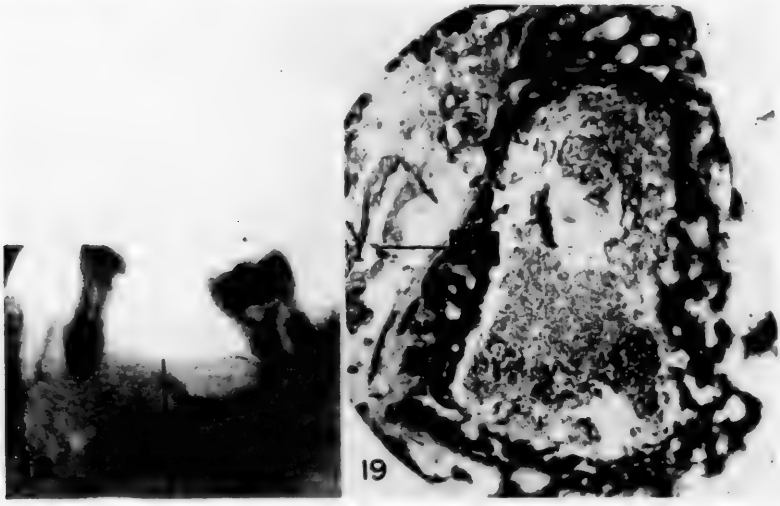

FIG. 18. Distinct outline of a cyst in the bone. Section indicated by vertical line is shown in *fig. 19*.

FIG. 19. Pathologic bone condensation around a cystic area. Section indicated by vertical line in *fig. 18*. High magnification of *I* is shown in *fig. 20*.

3. Condensation of the jaw bone due to *change of function* after extraction of teeth. The forces upon the jaw bone change from bending stresses of normal mastication to shearing stresses, which is seen in its least complicated phases in cases where posterior teeth have been lost and anteriors are still in place and functioning (*fig. 9*). Dense bone is deposited in the center of the jaw bone (*fig. 10*) instead of the outer border (*fig. 11*). This change, due to mechanical conditions, exhibits a similarity to the structure in the femur neck, which receives mainly shearing stresses (*fig. 12*) as compared with the shaft,

FIG. 20. Pathologic bone condensation. Haversian canals and systems can hardly be distinguished; the bone represents a mass of irregularly arranged bone layers. High magnification of *I, fig. 19.*

which receives bending stresses (*fig. 13*). This is in accordance with engineering laws, where in one case supporting material would be distributed near the neutral axis; in the other, as far away from it as possible (*fig. 14*).

FIG. 21. Periapical rarefaction and condensation, showing distinct outline. Section through *I* is shown in *fig. 22*.

FIG. 22. Pathologic rarefaction and condensation around periapical infection. *A*, necrotic material. Section through *I*, *fig. 21*. High magnification of *I* is shown in *fig. 23;* of *II*, in *fig. 24;* of *III*, in *fig. 2*.

4. *Rarefied areas* due to local but not necessarily pathologic conditions. After tooth extraction, the sockets do not always fill in to the same density as that of the surrounding bone. This may sometimes be due to inflammation, but frequently is due only to lack of normal stimulation. In the non-pathologic case, the rarefied area merges gradually into the surrounding bone (*fig. 15*). As may be seen in

histologic sections, the part filled in last is in the center of the alveolar process, which causes the appearance of a rarefied area in the x-ray picture (*figs. 16, 17*).

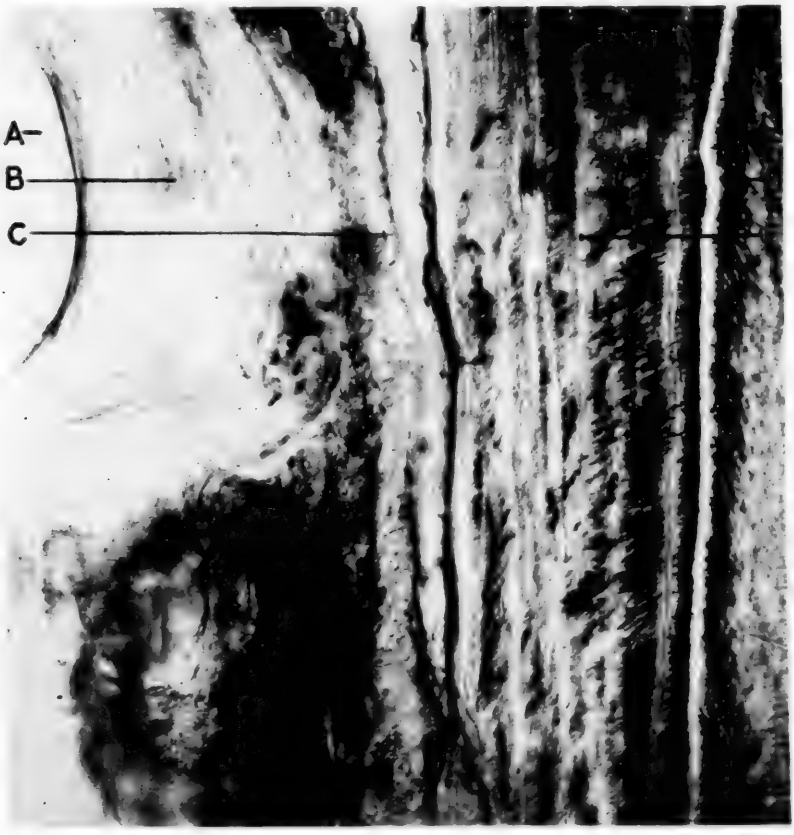

FIG. 23. Part of alveolar bone near cyst. *A*, haversian canal; *B*, haversian bone layers; *C*, subperiosteal bone layers; D, dentin; *E*, cementum. High magnification of *I*, *fig. 22*.

III. PATHOLOGIC CHANGES

1. *Cystic conditions.* The infected cyst presents the most active and probably the most effective attempt of the tissues to enclose the

infective focus. The bony wall shows a distinct demarcation in the x-ray picture (*fig. 18*), and it gives the sharply defined microscopic view of pathologic bone (*figs. 19, 20*).

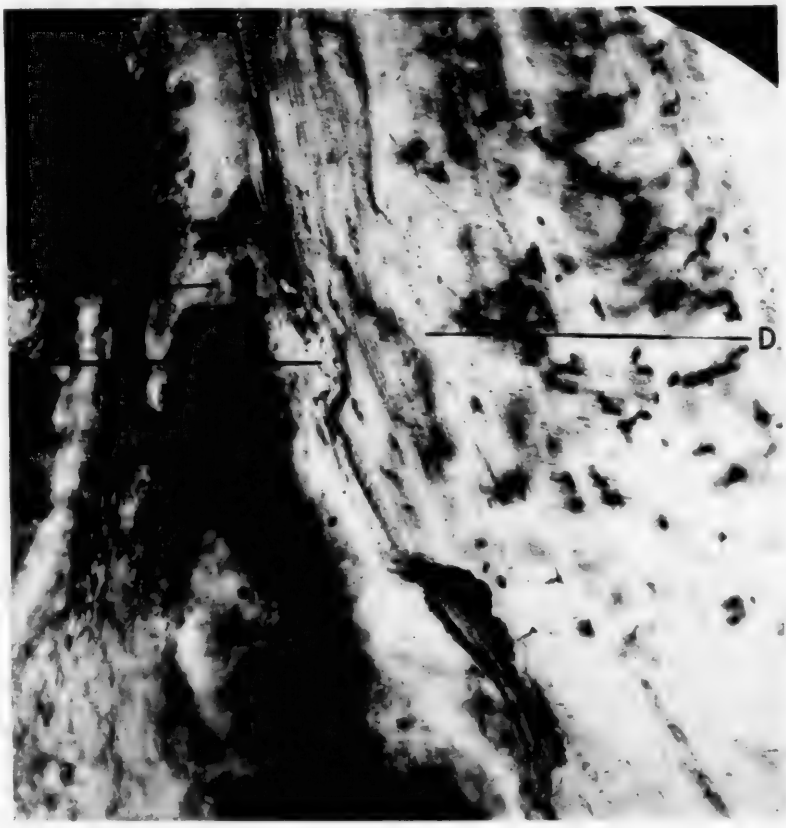

FIG. 24. Periapical infection. *A*, bone in a state of resorption; *B*, necrotic and infective material; *C*, resorption of cementum; *D*, cementum. High magnification through *II*, *fig. 22*.

2. *Periapical areas of infection* usually show some rarefaction within a zone of dense bone marking the outer border of the infected area (*fig. 21*). There are many gradations in the density of periapical bone

FIG. 25. Pathologic bone condensation around embedded root, with distinct outline. Section through *I* is shown in *fig. 26*.

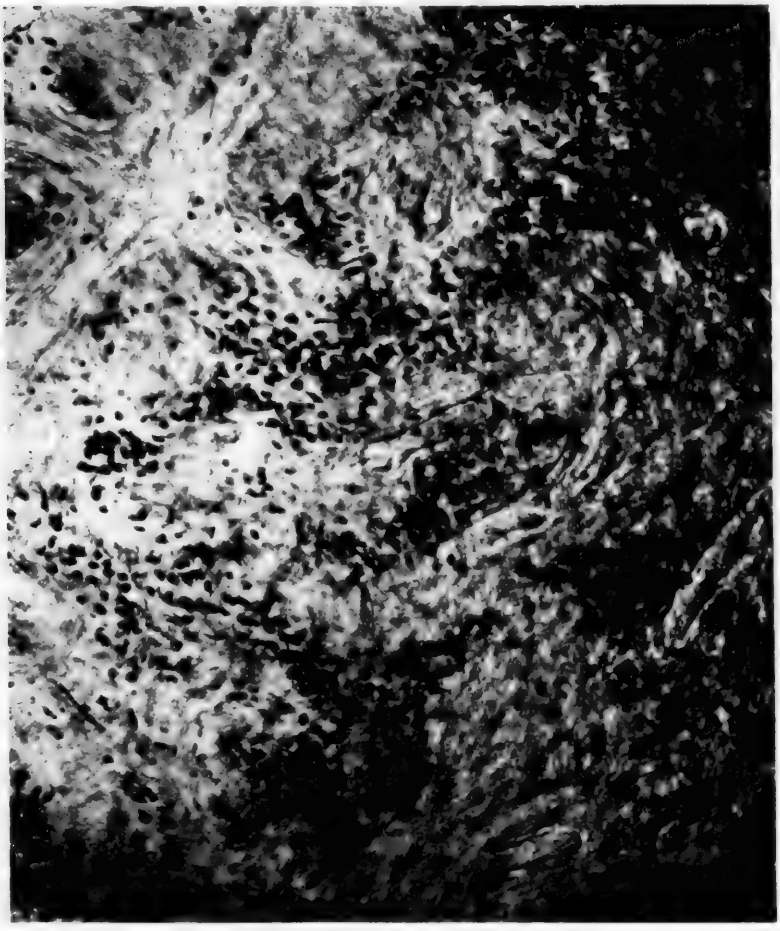

FIG. 26. Pathologic bone. Haversian canals and systems are irregular; the bone layers cross each other in a network in which there are remnants of cement and cement cells (dark pigmented corpuscles). Section through *I*, *fig. 25*.

as seen in the radiogram; the histologic picture is always that of infected bone (*fig. 22*). In the parts of the alveolar bone near the crest, some of the haversian systems are covered on the outside by undisturbed subperiostal layers (*fig. 23*). Nearer the apical area, where resorption of the subperiostal layers has occurred, resorption of the cementum may be beginning (*fig. 24*).

3. *Embedded roots* are sometimes clearly indicated by persistence of the peridental membrane, but in many instances areas of abnormal density may or may not be interpreted in the x-ray as root fragments (*fig. 25*). It has been shown that such bodies give evidence of being the remnants of infected root tips where dense bone has formed a wall around the infected area. Tightly interlaced bone lamellae have gradually grown into the root remnants, the infected parts of which have been broken down and carried away (*fig. 26*).

In conclusion, it should be stated that there is a distinction between non-pathologic and pathologic bone changes, and the nomenclature used to indicate these changes should definitely suggest the essential nature of the process. Since the term "osteitis" implies inflammation or infection, it should be reserved for pathologic conditions only, whether condensing or rarefying, and should not be used to indicate effects that result from changes in function. The term "condensing osteitis" is often applied to bone conditions induced by changes in function. In these cases the term "bone condensation or rarefaction" more correctly designates the condition.

The author wishes to acknowledge the assistance given him by Dr. John Oppie McCall in the studies on which this paper is based.

551 Fifth Avenue

TISSUE REACTION IN THE APICAL REGION TO KNOWN TYPES OF TREATMENT[1,2]

JAMES ROY BLAYNEY, B.S., M.S., D.D.S.

Chicago, Ill.

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I. INTRODUCTION

The purpose of this study is twofold: first, to observe the tissue reactions that occur within the pulp canal and about the root-ends of teeth following removal of the dental pulp. Second, to determine by histopathological research what form of clinical procedure gives the highest percentage of successful root-canal operations. Many dentists and physicians have questioned the advisability of pulp removal and retention of pulpless teeth. Some have taken the position that all such teeth should be extracted, even though the clinical and radiographic examinations are negative. Hayden (5) has stated that "pulpless teeth that are apparently negative in the radiogram may often harbor a sufficient number of bacteria to be a factor in systemic disease." Appleton, Rosenow, and Meisser appear to be of the same opinion. This is not a surprising statement when we consider the difficulty of removing a tooth from its socket without bacterial contamination. Since the study of root-end conditions from

[1] A dissertation submitted to the Graduate Faculty of the University of Chicago in candidacy for the degree of Master of Science, in 1928.

[2] Read at a meeting of the New York Academy of Dentistry, Hotel Commodore, New York City, January 24, 1929.

the bacteriological side is definitely limited by the possibility of contamination, I have studied the cellular reactions in these tissues in an effort to determine whether all root-ends of pulpless teeth are pathologic.

II. LITERATURE

In reviewing the literature of this subject one is impressed by the fact that most of the histologic study has been made of teeth gathered in the extracting room, where it was impossible to obtain an adequate history of the conditions that existed prior to, or the manner in which the case was handled during, treatment. Davis (1), in his study of many partially filled root-canals, found deposits of an osteoid material about the periphery of the canals. All his sections were prepared by grinding and were stained to show only the calcified structures, hence the soft tissue within the canals could not be studied. Grove (2) reports as follows: ". . . . if the pulp tissue is not all removed, a transformation or a reorganization of that tissue occurs and fibrous tissue is formed to take the place of the pulp. Cementoblasts then migrate into the canal and form cementum to furnish attachment for this tissue, when the formation of cementum stops, at least to a degree." He concluded that the underfilled canals have a better prognosis than those filled to the apex, because there is less destruction of the apical tissue, and that there is probably a greater opportunity for infection where the foramen is completely opened.

Hatton and Skillen (4), in discussing the changes that occur in this tissue, describe first a resorption, which may extend into the dentin; second, a process of repair-calcification quite similar to that reported by Davis; and third, a stage in which calcification ceases, which they term the resting period. Hatton (3) also suggests that the apical portion of the dental pulp reacts differently from that of the middle or coronal portion, and that the fibrous-tissue framework and the cementum-producing cells may resist insults better than the odontoblasts, pulp cells, and nerves. Skillen (8), in discussing this subject, has called attention to the fact that histologic evidence bears witness to the fact that many teeth have been carelessly treated. He concludes that the burden of failure does not rest upon the tooth alone, and that proper treatment consists in the reduction of the pulp tissue

to the smallest amount possible without too great an injury. However, he has not attempted to offer a definite program of treatment as a guide for those engaged in operative practice.

III. METHODS USED IN THIS STUDY

Believing that an accurate clinical record should be available when the histologic study of the root-end is made, I began in 1918 the accurate recording and filing of the histories of all the cases that came under my observation. To date several thousand such histories have been collected. From this number I have examined microscopically approximately 250 teeth, some of which were extracted shortly after treatment owing to immediate failure; others remained in place several years after pulp removal and were extracted on account of conditions in other parts of the jaw necessitating restoration by means of a full denture.

Whenever the teeth were to be extracted a radiogram was taken to secure a knowledge of the condition of the root-end, and notations were made concerning the overlying soft tissue, the occlusion, and whatever else was thought to have a bearing on the case. As a fixing agent, both 10 per cent formalin and Zenker's solution were used. The hard tissues were decalcified in 4 per cent nitric acid and then washed in running water for about three days. The celloidin method of embedding was employed in all cases. It has been my experience that, to ensure thorough penetration of the embedding material, the tissue should remain in thin solutions of celloidin as long as possible, in some cases several months. For study of the soft tissue, staining with dilute hematoxylin (Delafield's) and eosin was used; for the hard tissues and repair-calcification, thionin and picric acid were used as described by Hatton (3).

IV. HISTOLOGIC FINDINGS

Very early in the study I observed that it was unusual to find a root-end with but a single apical foramen. Grove (2) and others have nicely demonstrated that the soft tissue within these apical ramifications is not dental pulp, but an invagination of the periodontal membrane. There was for some time a general belief among practitioners that pulp removals could not succeed in the presence of

multiple apical openings. However, it is my belief that the accessory canals provide an abundant collateral circulation that aids in the repair

FIG. 1. Longitudinal section through tooth and mandible. Note principal pulp canal at *A*, and the ramifications within the apical cementum, *B*, *B*, *B;* cementum at *C;* dentin at *D;* and peridental membrane at *E* (about 50 ×).

processes following pulp operations (*fig. 1*). If their existence is recognized, and the operator plans his procedure so as to leave this tissue

uninjured, the presence of such apical openings is an aid rather than a hindrance to pulp-canal therapy.

Another observation of importance to practitioners is my finding that, regardless of how completely the canals appeared to be blocked at the time of operating, the root-end was never entirely closed.[3] To the clinician this should mean that, even in the extremely fine and apparently fully calcified canals, the same precautions must be observed to prevent infection and chemical necrosis of the soft tissue as are used in cases with accessible canals.

To illustrate the repair processes that occur after pulp removals, I have arbitrarily made two groups of the cases observed: those with radiographically normal root-ends, and those in which the radiogram indicates that a definite root-end change followed treatment. From the first group two typical cases have been chosen. In one, the tooth was extracted for microscopic study seven months after treatment, while the other was allowed to remain in the jaw five years before removal. The plan of clinical treatment in the first case was as follows: The pulp of a maxillary molar was carefully examined and found to be clinically normal. The region was anesthetized by injecting 2 cc. of 2 per cent solution of novocain and 1–100,000 solution of adrenalin along the posterior superior branch of the maxillary division of the fifth cranial nerve; also 1.5 cc. over the upper second bicuspid. A rubber dam was applied and the entire field of operation painted with 7 per cent tincture of iodin. Using sterile instruments, the pulp chamber was exposed, the orifice of each canal brought into full view, and the pulp tissue amputated at a point thought to be within the apical third of the canals, which were washed with sterile, normal, salt solution to remove all particles of dry blood. After the canals had been thoroughly dried, a dressing moistened with eugenol was placed in each and the tooth sealed with cement. Four days later the tooth was opened to curette and enlarge the canals, to place the filling material, and to insert diagnostic wires for the purpose of x-ray measurement. Then another dressing of eugenol was applied. Later the surface of each canal was slightly moistened with eucalyptol, and filled by packing short sections of gutta-percha cones. Radiographic ex-

[3] Hatton, however, has reported seven cases in which all foramina were completely calcified.

amination showed all canals to be obliterated by a solid homogeneous mass of filling material as far as the junction of the middle and apical thirds of the root. At the time of extraction, radiographic evidence indicated that the root-ends were normal.

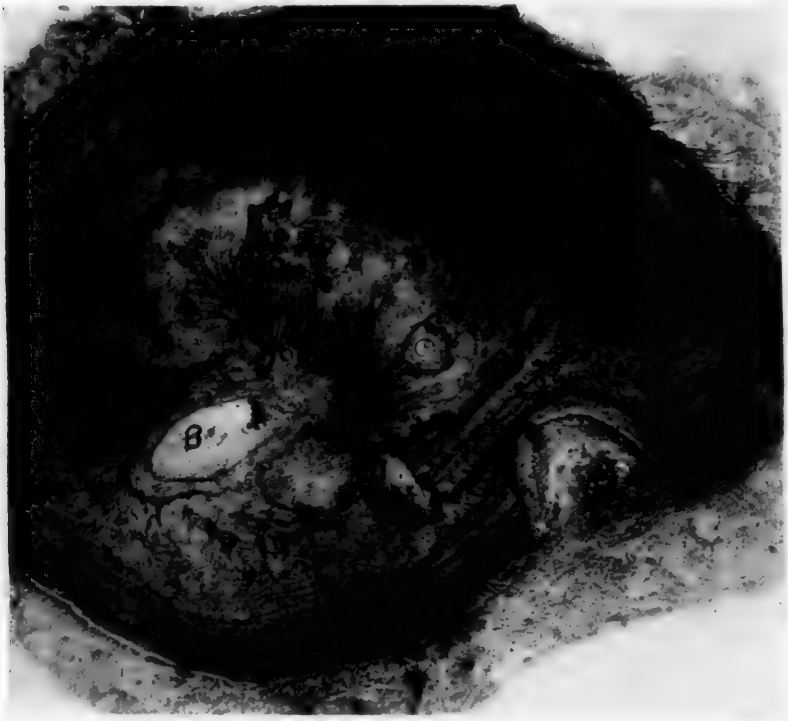

FIG. 2. Apical region. Note presence of canals at A, B, C, D, E, each showing evidence of resorption repaired by deposit of cementum (about 35 ×).

Histologic study of the apex of the lingual root revealed five distinct foramina, each containing a fibrous tissue entirely free from inflammatory elements. In *fig. 2*, foramen *A* is shown opening to the exterior. Within its lumen is a soft tissue not unlike periodontal membrane. In this foramen (*A*), as in the other four, are found definite records of a resorption in process of repair by the formation of

calcified material resembling cementum. It is thought that this absorption of the canal wall, as well as of soft tissue that has become necrotic owing to injury at the time of operation, occurs during the period of active inflammation, which always follows operative procedures. The single layer of cells, with large deeply staining nuclei

FIG. 3. Section at a level slightly removed from the apex in the gingival direction. (See *fig. 2*). Canal *A* now entirely closed. Note cells *B*, which resemble cementoblasts, and rim of cementum deposited after absorption. No evidence of inflammatory elements found within soft tissues (about 160 ×).

completely surrounding the lumen of all the canals, may be traced into the periodontal membrane in foramen *A*. These cells are undoubtedly cementoblasts, which have migrated into the canal and are active in effecting the repair. In *fig. 3*, the contents of canals *A* and *B* are free from inflammatory elements, and in *B* a calcified

deposit resembling cementum was formed upon dentin. *Fig. 4* illustrates the fusion of canals *A* and *B* to form the large principal canal. Here again we see the record of resorption: in certain areas, there is deposition of a cement-like substance upon the dentin; in other areas, the cells are in contact with the dentin, ready to lay down their de-

FIG. 4. Union of canals *A* and *B* shown in *figs. 2* and *3*. Notice character of soft tissue within canal. Resorption at *R, R, R,* with repair calcification at *C, C, C,* and cells forming cementum at *D, D, D* (about 140 ×).

posit of repair calcification. The gutta-percha filling is shown in *fig. 5*. Note that the process of resorption ceased before this level had been reached, and that the full diameter of the canal is entirely obliterated by the filling material.

The conditions involved in the second case, and the clinical treatment, closely parallel those of the first with two possible exceptions:

(a) this pulp was exposed by dental caries and therefore was not normal, and (b) a small amount of chloro-percha was used as a filling material with short sections of gutta-percha cones.

The microscopic study of the lingual root-end shows only one rather large foramen (*fig. 6*). Here as in the previous figures we find a record of an extensive resorption (*A*), which has been repaired by a

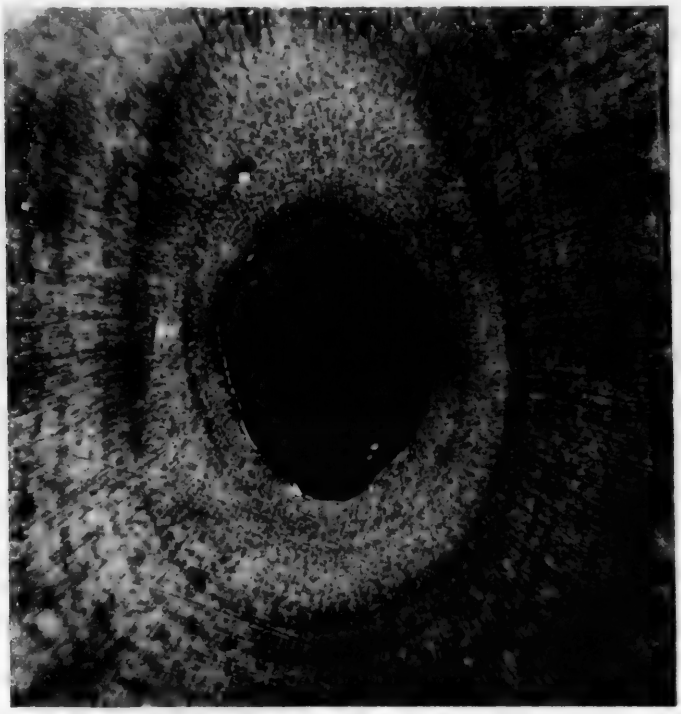

FIG. 5. Base plate gutta-percha root filling in canal (about 40 ×).

deposit of bone cementum (*C*). The soft tissue closely resembles periodontal membrane, and is entirely free from all evidence of inflammation. In *fig. 7*, which is taken from a level slightly nearer the gingival region, we find the line to which resorption progressed (*A*); the repair calcification (*C*), which has materially reduced the size of the canal; and fibrous tissue (*F*) within the lumen. At two points

(*E*) there are crescent-shaped masses of gutta-percha. It will be noted that the gutta-percha has been completely surrounded by calcified material as if nature wished to wall it off from the soft tissue within the canal. More will be said in another paragraph concerning the reactions that occur about root-filling materials.

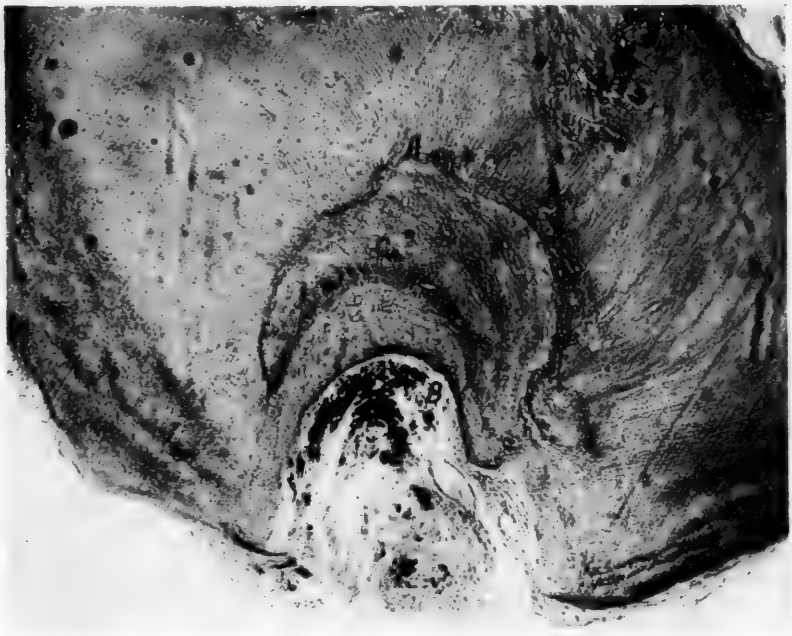

FIG. 6. Apex of lingual root. Line to which resorption progressed, *A;* repair by bone cementum, *C;* "formalin mud," *B* (about 100 ×).

As one studies sections progressively nearer the mid-root (*figs. 8, 9, 10*), it is noticed that the amount of resorption along the lateral surface of the canal and the amount of calcified material become progressively less. It is quite evident that, following the resorption process, the soft tissue within the canal (and surrounding whatever filling material may be present) is no longer granulating but fibrous in character. This study also seems to indicate that both the resorption and the repair processes begin at the apex; and that, as the

FIG. 7. Resorption progressed to *A;* repair by bone cementum, *C, C, C;* dentin, *D;* gutta-percha at *E, E,* which has been entirely surrounded by cementum. Fibrous tissue remaining in canal at *F* (about 100 ×).

FIG. 8. Resorption to *A;* bone cementum, *C;* dentin, *D;* gutta-percha, *E, E;* fibrous tissue within canal, *F*. Note that total amount of cementum gradually diminishes as distance from root-end increases.

FIG. 9. Root canal much enlarged by resorption of dentin, *A;* small amount of repair calcification, *C;* gutta-percha cone surrounded by fibrous connective tissue, *F*. Note abundant blood supply to connective tissue, which is ready to begin calcification at *G, G, G* (about 100 ×).

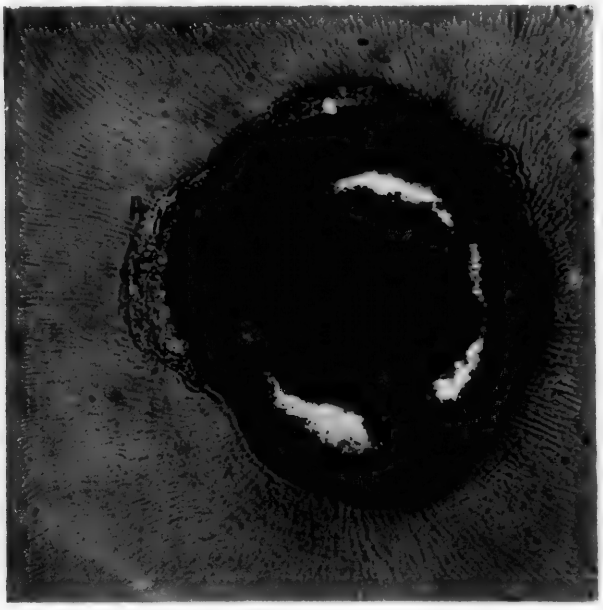

FIG. 10. Some resorption at *A;* space left after resorption filled by well organized connective tissue, *F*. Repair calcification ready to begin (about 100 ×).

FIG. 11. Section from buccal root. Extent of resorption, *A;* repair calcification, *C*, *C;* plugs of fibrous tissue, *F*, *F;* dentin, *D* (about 125 ×).

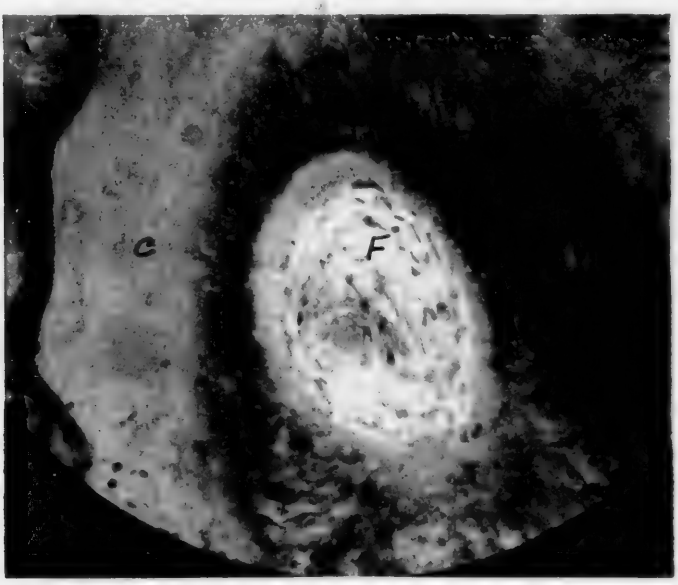

FIG. 12. High magnification of buccal root, showing character of calcification, *C;* soft tissue within canal, *F* (about 250 ×).

obnoxious material is removed and the granulations are replaced by fibrous tissue, the cementoblasts of the periodontal membrane migrate into the apical opening, gradually extending down into the canal along the dentinal wall. The buccal roots exhibit the same type of reactions (*figs. 11–12*).

FIG. 13. Granulation tissue and portion of apical cementum. Note that soft tissue lies in physiologic relation to cementum. (See *fig. 14* for high power of X).

For the illustration of some of the retrogressive changes in the periapical tissue that occur after the removal of a vital pulp, we have chosen a lower molar with a definite involvement surrounding one root-end. The pulp was removed eighteen months before extraction,

following the same general plan described for the former cases. The distal root-canal was cleaned and filled very close to the root-end, but the mesial root was hardly opened, the filling reaching into the canal only a few millimeters. At the time of extraction, a mass of soft tissue 4 mm. in diameter was attached to the mesial root, while the distal end appeared normal. The roots and soft tissue were retained

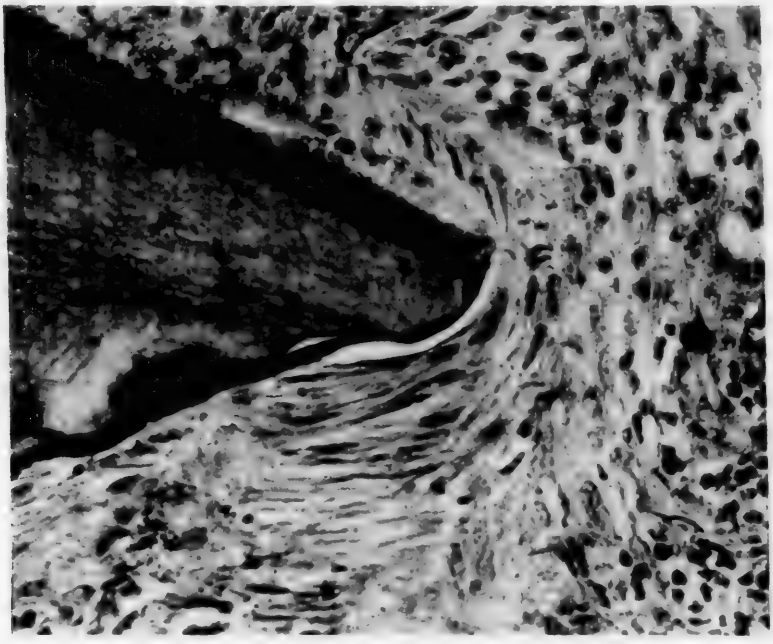

FIG. 14. High magnification of *X, fig. 13.* Note relation of soft tissue to cementum.

intact, in order to study the relation of the changes in the periapical tissue to the conditions found within the pulp canal. The specimen was sectioned in stages or steps, so that I might have a fairly accurate idea as to the level that each section represented.

The first group of sections, which are from the extreme apical portion, show that the periodontal membrane of the mesial root was replaced by a mass of tissue consisting of many mononuclear leucocytes

and plasma cells, a few polymorphonuclear leucocytes, fibroblasts, and blood vessels (*figs. 13–14*). The cementum shows many areas of resorption, also areas disclosing the attachment of bundles of fibers. It is evident that at all points the soft tissue lies in physiologic contact with the cementum, and that at no point is the cementum denuded. At a slightly higher level (*fig. 15*), there is a large area of resorption,

FIG. 15. Extensive area of resorption, *A*, reaching into pulp canal, *P*.

which extends into the root and unites with the pulp canal. Both the resorption area and the pulp canal are filled with the type of tissue that surrounds the root-end. At a somewhat higher level the pulp canal is being enlarged by irregular resorption areas, and the character of the soft tissue has been materially changed, polymorphonuclear leucocytes prevailing. Just beyond this level the canal contains ne-

crotic pulp tissue, debris, and some gutta-percha filling. It is interesting to note that none of these retrogressive changes were found in the distal root. This evidence seems to indicate clearly that the mesial canals were not properly treated. The pulp tissue left in the anterior

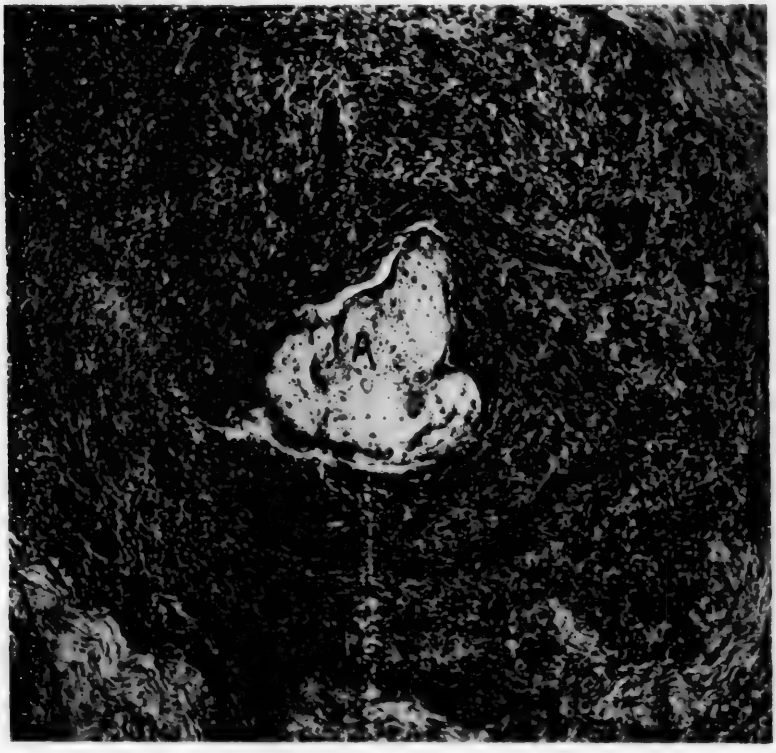

FIG. 16. Portion of base-plate gutta-percha, *A*, which is surrounded by mass of polymorphonuclear leukocytes, *B*.

canals did not possess the power to repair itself after the trauma caused by the treatment, so nature gathered these forces in an attempt to remove the irritating material.

Many such apical granulomas result from continued low-grade irritations, caused either by toxic material within the root-canal, by

mechanical trauma, or by chemical action. The formation of a granuloma is a defensive reaction of the soft tissue surrounding the root-end. The dark area surrounding a root-end, as shown by the radiogram, is produced by expansive growth of this soft tissue.

FIG. 17. Periapical tissue, containing gutta-percha, *A*, spicule of dentin, *B*, apical cementum, *C*, two and one-half years after root filling. Note attempts of resorption and partial encapsulation of gutta-percha; also resorption of spicule of dentin and cementum.

Plasma cells, which are often found in great numbers, are slightly differentiated from the group of mononuclear wandering cells. It is thought that they are not phagocytes, but that they produce enzyme which is active in resorbing certain dissolved toxins or products of

disintegration, and of disposing of them intracellularly (6, 7). In the therapeutic treatment of such cases, the pathological processes involved must be kept constantly in mind. During treatment this inflamed area must not suffer additional injury by mechanical or chemical trauma. If the formation of a granuloma is a defensive process, the aim of the therapeutist should be to remove the exciting

FIG. 18. Mass of gutta-percha, *A*, within periapical tissue, which is completely encapsulated in dense fibrous tissue, *F*. Gutta-percha within canal, *B*, surrounded by extensive repair calcification, *C*.

cause by a technic that is based upon surgical principles, and then to leave the absorption and repair to the biologic forces in the patient. In many of the cases in which there is a physiologic relationship between the granulation tissue and the cementum, the inflammatory elements gradually give way to the formation of fibroblasts that are active in effecting the repair following thorough cleansing, treatment, and filling of the root-canal.

V. ROOT FILLINGS

Ever since the earliest attempts it has been considered good practice to fill the root canal after removing the pulp. Many different materials have been popular, but there has been little effort to study the ensuing cellular response. In this report the only filling material used in the cases studied was gutta-percha, either base plate or a combination of gutta-percha and chloro-percha. For years there has been an active controversy on the danger of carrying filling material into the periodontal membrane. Clinically it is a well known fact that a large percentage of teeth with over-filled canals develop radiolucent areas about the apices. Also that shortly after such a filling is placed, the tooth becomes quite sore, and in some cases elongated and even loose, depending upon the amount of over-filling. In the histologic study of such a case, which was removed one week after filling, I found the gutta-percha completely surrounded by polymorphonuclear leucocytes, as if the tissue had been trying to throw off this foreign body (*fig. 16*).

In other cases, in which gutta-percha had passed into the periodontal membrane, the time between the filling and extraction of the teeth was much longer. In these cases it seems that, following the acute reaction just described, there was an effort to remove the root filling by resorption (*fig. 17*), and that, failing in this, the entire mass of gutta-percha was completely encapsulated in dense fibrous connective tissue (*fig. 18*). While this is the exact course of events we might expect, following the introduction of a non-absorbable foreign body, only a small percentage of cases have such a favorable termination. Of these studied, only one shows complete healing. The others show a variable amount of residual granulation tissue, with only a slight tendency to encapsulation by fibrous tissue. In the case studied five years after treatment, the apical portions of the root-filling material were entirely encapsulated by calcified material, while deeper in the canal the filling was surrounded by fibrous tissue. Throughout the entire study I found that the classical foreign-body reaction prevails, although complete healing is accomplished in relatively few cases.

VI. CONCLUSIONS

The evidence gathered from this study strongly suggests the following conclusions.

(1) The dental pulp may be removed without causing irreparable damage to the periapical tissue, provided the following definite plan of operation is carried out: (a) surgically clean technic; (b) use of only mild antiseptics, all caustics being eliminated; (c) avoidance of injury to soft tissue in apical foramina; (d) removal of all true pulp tissue; (e) filling of the canal with a bland, non-irritating, non-absorbable material to near the site of amputation.

(2) Following the operation there is begun, in the apical region, a process of resorption that enlarges the apical foramen, or a new channel may be cut that more successfully meets the conditions within the canal.

(3) These resorptions may heal, with the formation of calcified material resembling cementum.

(4) Many of the apical foramina may be reduced in size by repair calcification. But this reduction seldom obliterates the canal, as sufficient space usually remains for an efficient circulatory apparatus.

(5) The filling material, when in contact with soft tissue, excites a foreign-body reaction. Better results are obtained in the cases that are slightly underfilled.

In drawing conclusions from the microscopic study of each case, the clinical diagnosis, the methods of treatment employed, and other clinical data of importance were always considered.

The writer wishes to acknowledge his indebtedness to Drs. H. Gideon Wells and Frederick B. Noyes for their assistance and many valuable suggestions.

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DISCUSSION

E. G. Van Valey, D.D.S. (New York City): Dr. Blayney has presented to us, in the paper to which we have just listened, results obtained in a study extending over a decade. To-night Dr. Blayney is the teacher, and we the students. My discussion is undertaken in that spirit.

We were invited to consider not methods of treatment of pulpless teeth, but the reactions of tissues in the apical regions of teeth that had been treated; and, incidentally, to choose a form of treatment from those studied which promises to give the best percentage of successful results.

All the cases presented were of teeth in which the pulps had been removed. In some cases the pulps were vital and apparently not diseased; in others they were in various stages of disease. Some of these cases must have been infected, but unfortunately no tests seem to have been made to determine the organism causing the infection, nor to differentiate the types of reaction that might be caused by various microörganisms.

Teeth that show definite signs of bone change at the apex before treatment have not been taken up in this paper. Therefore we are given no enlightenment regarding tissue reactions at the apex of these baffling cases.

We were shown slides of teeth in which there was an excess of gutta-percha in the apical tissues. The reaction of nature, we were told, was "a foreign-body reaction," but the slides show a typical inflammation such as might be caused by infection. Was the gutta-percha sterile? Was it forced into an area which still harbored infection? Without the answer to questions such as these our conclusions concerning the reactions that follow the complete filling of the apical foramen must of necessity be faulty.

The slides that were shown have again demonstrated the fact that new calcified substance, possibly somewhat similar to cementum, may be deposited along at least parts of the canal walls in teeth from which the pulps have been removed. This formation of calcified substance seems to be similar to that laid down in other fibrous tissue, as for instance in a tubercular nodule in the lung. The blood supply to the involved part seems to be an important factor in the deposition of this new calcified material. In the book on "Physiology of bone," by Leriche and Poligard,

we find the following statement: "If by any process whatever the activity of the circulation is increased in the vicinity of a bone, the latter becomes rarefied." Conversely, the reduction of circulation tends to produce a condition favorable to calcification.

Dr. Blayney referred to the work of Drs. Grove, Hatton and Skillen. In their studies attention was called to the fact that although the laying down of calcified material had been started, this process was not continued until the apical foramen was filled. It is important to note that the apical foramen is not closed by this repair calcification.

Dr. Blayney points out that he has never found a tooth in which the foramen was completely obliterated, no matter how fine or impenetrable the

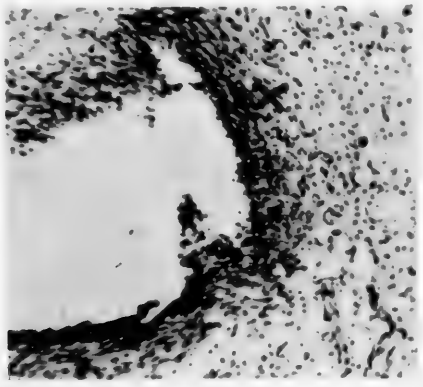

FIG. 19. Pulpal abscess; *X*, deeply staining cells typical of inflammatory reaction.

canal seemed to be. Again, in one of his conclusions he states: "Many of the apical foramina may be reduced in size by repair calcification. But this reduction seldom obliterates the canal," etc. This conclusion removes the comforting probability that the root-end will be closed with a normal layer of cementum.

The first case presented in Dr. Blayney's paper was that of a molar with a normal pulp that had been surgically exposed and removed, and the canals filled to a point within the apical third. Seven months later the tooth was removed and sections made. Two sections near the apex show very clearly the repair calcification progressing along parts of the canal walls. The third section (*fig. 4*), further from the apex, still shows some new calcification, which however does not surround the periphery of the canal so completely. In addition the picture seems to show an inflamed area. In

fig. 20 I have photographically reproduced that part of Dr. Blayney's section. Before taking up the study of this illustration, however, let us examine *fig. 19*, which, made by Dr. Lester R. Cahn, is a section of a pulpal abscess. Note the degenerated abscess cavity surrounded by large deeply staining cells (*X*), which are typical of inflammatory reaction. I show this photomicrograph as a basis for comparison with *fig. 20*, where we see again an open space, surrounded by large deeply staining cells (*X*), which strongly resembles the pulpal abscess in *fig. 19*. In the history of this tooth (*fig.*

FIG. 20. Open space, surrounded by large deeply staining cells, *X*, which strongly resembles the pulpal abscess in *fig. 19*. *C*, new cementum-like substance.

4), as outlined by Dr. Blayney, the pulp of a perfectly normal tooth was surgically exposed, and later the canal was filled to a point within the apical third. Seven months afterward he made the section in *fig. 4* (*fig. 20*). Dr. Blayney points out that no inflammatory processes were apparent. I feel that although new cementum-like substance may have been forming, such as that in *C*, in the reproduction of *fig. 4* (*fig. 20*), this pulp remnant was already inflamed and probably would have died if given a longer period of time.

The more tissue left at the apex beyond a root-canal filling, the greater the chance of ultimate degeneration of that tissue. This is brought out in

the second tooth selected by Dr. Blayney (page 234). The root-canal filling in the mesial root of a lower molar extended only "a few millimeters" into the canal (*figs. 13–15*). At the time of extraction, eighteen months later, the section showed that there was not only inflammation and necrosis within the unfilled canal, but that a typical granuloma had formed at the apex. I agree with Dr. Blayney that this canal was surely improperly filled. I believe that if this canal had been filled under favorable conditions as was the distal canal, that is, almost to the apex, no pathologic change would have taken place at the apex.

It would be well always to remember this statement in Dr. Blayney's paper: "The aim of the therapeutist should be to remove the exciting cause by a technic that is based upon surgical principles, and then to leave the resorption and repair to the biologic forces in the patient." It is this,

FIG. 21. Radiogram of a tooth filled sixteen years ago

after all, that effects the cure. But will these biological, curative processes remain strong enough permanently to safeguard an unfilled apex? The usefulness of a root filling does not end when a degenerated apical area is cured. It must remain cured even during a period of lowered resistance. Such a condition of lowered "biological forces" may never arise, but there is always the possibility that impairment of normal resistance may occur after a prolonged period of comparative immunity. It is during such a period of lowered resistance that many teeth with fillings almost to the apex become a focus of infection.

Fig. 21 shows a radiogram of a tooth filled sixteen years ago. Here the filling is about two-thirds of the way to the apex. Only during the last month have any signs of a focus of infection been present. I feel that had this apex been filled sixteen years ago, the present reinfection would not have occurred.

FIG. 22. *A*.—Radiogram showing granuloma at end of second bicuspid (Nov, 4, 1911). *B*.—Same tooth, showing regeneration two and one-half years after treatment (May 18, 1914). *C*.—Same tooth, showing regeneration nine and one-fourth years after treatment (Feb. 11, 1921).

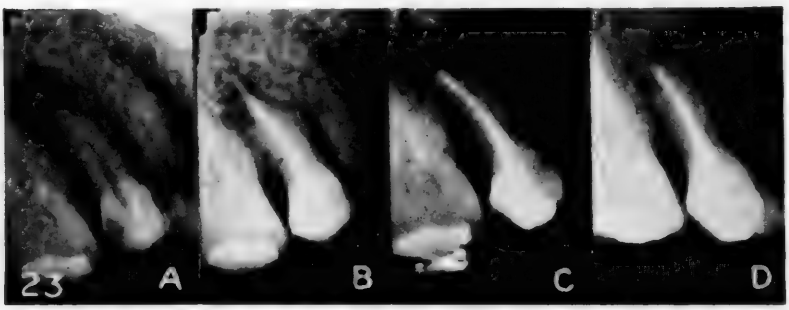

FIG. 23. *A*.—Radiogram showing original condition, Jan. 1912. *B*.—Same, May 1912. *C*.—Same, Feb. 1919. *D*.—Same, Dec. 1920.

FIG. 24. *A*.—Radiogram showing the original area of bone degeneration. *B*.—Radiogram showing the root-filling and large mass of excess gutta-percha. *C*.—Radiogram showing that bone has completely filled the degenerated space.

Let me refer now to conclusion 5. I quote from Dr. Blayney's paper: "The filling material, when in contact with soft tissue, excites a foreign-body reaction. Better results are obtained in the cases that are slightly underfilled." Are we to understand that the end of the root filling is not in contact with soft tissue? Then with what is it in contact? No matter where the root filling might end, it would still be in contact with soft tissue of some variety. I feel that there is less danger in having the end of the root filling in contact with the soft tissue at the apex. With the soft-tissue contact in this position, the "biological forces" would have a better opportunity to keep the apical tissues healthy when the bodily resistance may be lowered, than if all the action took place in a remnant of an unfilled root canal.

The three cases which follow, although not examples of ideal root-canal filling, illustrate the fact that nature takes care of rather large areas of bone degeneration, if the apex is closed with filling material. *Fig. 22* shows radiograms taken in 1911, when the tooth was first treated; also in 1914, and again in 1921. The bone tissue remains normal. In *fig. 23* we see the original condition in 1912; also radiograms taken in 1919 and 1920. The last radiogram we have been able to secure, taken in 1926, shows the tissue still normal—a record of fourteen years. In *fig. 24* the original area of bone degeneration is shown; then the root filling, with a large excess of gutta-percha. The last radiogram shows that bone has completely filled the degenerated space. I wonder what the reaction in the apical area would have been if these canals had been filled "almost to the end of the root."

The brief study of this paper that I have had the opportunity of making has only stimulated my desire to learn more under the teaching of Dr. Blayney.

Dr. Blayney: Dr. Van Valey raised the question as to why the discussion was confined to the removal of vital pulps. Why did we not discuss the tissue reactions that occur in the treatment of diseased root ends? We might divide this big subject into two divisions: the anterior problem and the posterior problem, if I may call them such. It seems to me that the important problem for us all, as practising dentists, is to learn how to remove a pulp from a tooth that has a normal root-end and to keep that root-end normal. When that time comes, we shall not be called upon to treat nearly so many cases having root-end involvement. True, in the past the profession has been quite concerned as to how such conditions are to be treated. Nevertheless, I still believe that that is the posterior problem. At some other time, I hope to take up the study that Dr. Van Valey has suggested, but it certainly could not be carried on as part of this study.

Dr. Van Valey asked: Was sterile gutta-percha carried through the root-end? My only reply can be that it was sterilized properly before we started to use it, but in a bacteriological sense I do not presume it was sterile. No surgical operation is ever carried out under conditions of sterility as recognized in a bacteriological laboratory. However, I can say that I believe the gutta-percha and the entire technique were surgically clean, and that is all we can hope for. In discussing these slides with some of my instructors, that very question was raised. I asked: "Do you believe you see in this particular slide any evidence of infection?" Upon close study we found no evidence of pyknosis, none of dying leukocytes, and none to indicate formation of an abscess. It seemed to be the consensus of opinion of these instructors that the acute polymorphonuclear reaction was an attempt of nature to reject—just as around any sterile foreign body. Maximow says that any sterile foreign body is surrounded by an inflammatory reaction. Later on that inflammatory reaction subsides and fibroblasts develop in an effort to encapsulate it.

I was very glad that Dr. Van Valey reproduced *fig. 4* and presented it to you. I wonder, Dr. Van Valey, if I might have the privilege of returning that slide to the screen (*fig. 20*)?

Dr. Van Valey: By all means, and I should like to ask a question about that particular slide. One of your diagrammatic sections[4] showed a small area of inflammation, and very close to this was a point at which new calcification was taking place. Do you think that the slide now on the screen (*fig. 20*), with a point of inflammation at *X* and another of new calcification at *C*, is similar to the diagrammatic slide I refer to?

Dr. Blayney (*in conclusion*): The other day, after the meeting of the Chicago Dental Society, I showed this slide to a visiting colleague, who examined it under the microscope and asked the question: "Is that inflammation?" I think you are justified in calling it an inflammatory reaction, but I think the cells are cut at cross-section rather than longitudinally, and that it is these very cells, the fibroblasts that Maximow describes, that are active in forming this tissue. I don't think that is an infected area. The section is rather thick. It is rather difficult to get sections thin enough to make a detailed study of cells. Maximow cuts his sections two to three microns thick. It is difficult to get a section of a tooth less than ten microns thick.

I wish to refer to the case I cited as a failure, with the development of an infected area at the root-end. As you know, in all of this work personal equations enter, and all students are not of the same caliber. When I found this area I was disappointed to see the tooth going bad. But when

[4] These schematic slides were not submitted for publication.—[Ed.].

I looked it up, and noted the record of all the clinical treatment, I really was not surprised. I do not doubt that that tooth was infected at the time of operation. The student had not entered the anterior canal, because they are difficult to enter, and I might also question his desire to carry out the treatment under a perfectly aseptic technique. The distal canal, easy to open, was successfully treated. Nature was capable of taking care of this region. But in the mesial canal too large a burden was placed upon the tissue for anything but failure.

A question has been raised about the point of amputation, as I term it. In the schematic slides[4] you saw that the pulp was amputated within the region of the dento-cementum junction. I believe that is the ideal point. However, we are not capable by any means of carrying that out in all cases. Sometimes we have to fall far short of our ideal, but from this study the fact stands out very strongly that that is the point toward which we should strive. Comparison of the success of underfilled canals with the success of overfilled canals (or canals that are filled to the end of the root), is very interesting but needs considerable further attention. However, from a study of several thousand cases (I believe we have 14,000 to 15,000 histories on file), I have found that we had better success in canals that were slightly underfilled. I do not mean, by "underfilled" canal, a filling going into the canal only one millimeter, or two millimeters, or three millimeters; I want the filling to go into the canal as far as we have been able to operate, and to operate into the canal to a point as near the dento-cementum region as can be approached mechanically. So our tabulations of a good many cases seem to show that we have had better success in cases that were filled just a bit short of the root-end than where the filling material was carried beyond the root-end.

The last question: Is the gutta-percha placed in direct contact with living tissue at the root-end? If the canal is filled with a measurement control to the point of amputation, I do not believe that the gutta-percha, when placed, comes in contact with a vital tissue. In the schematic slide you saw necrotic tissue, and inflammatory cells coming up in an effort to remove it. What then will happen if we put gutta-percha into living tissues? It will produce fresh necrosis—the tissue will be injured. The cells will die, and will have to be resorbed before the reparative processes can develop. I believe our ideal should be to carry the root-canal filling close to the site of amputation, but to avoid pressure upon the tissue in that region. Then as the healing process goes on, embryonic tissue, and later fibrous tissue, will surround the root-canal filling, and lie in contact with it without pressure.

THE BIOLOGICAL CONCEPT OF PARTIAL PROSTHESIS, AND THE TECHNIC FOR A FIXED BRIDGE THAT COMPLIES WITH IT[1]

W. G. BRIDGE, D.M.D.

Tufts College Dental School, Boston, Massachusetts

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I. INTRODUCTION

Dentistry is passing through a period of transition. Old ideas are being challenged. Educational institutions are more or less at sea as to the best course to pursue to insure intelligent growth for the future of dental education. In partial prosthesis many of the systems and methods in vogue for years are being questioned. In attempting to solve the many complex problems in the field of partial prosthesis, it is important to have a clear conception of their nature.

From the earliest beginnings, very little attention has been given to the biological character of adequate partial prosthesis. Practically all the text books on partial prosthesis are devoted principally to the mechanical phase of the subject. A study of the literature pertaining to this subject indicates that partial prosthesis is considered solely as a mechanical problem. In reviewing books on this subject, I note that in one, on crown and bridge work, fifteen of the eighteen chapters are devoted to technic. Thus it may be seen that the viewpoint is essentially mechanical. Methods, system, technic, have become the watchwords, the essence, of partial prosthesis. If the mouth were a plaster model or a piece of steel, an exclusively mechanical concept would have a rational basis to work from, but such is not the case. The very fact that we work on living tissue raises the partial prosthesis problem above the realm of ordinary mechanics. Our appliances

[1] Read at a session of the Seventh Annual Meeting of the International Association for Dental Research, Northwestern University Dental School, Chicago, Ill., March 23–24, 1929.

must be so constructed that they will substitute for, and function with, the vital structures of the mouth.

Inasmuch as we have been studying the problem from the mechanical viewpoint for the past sixty or seventy years, and are still far from attaining desired results, it seems reasonable and logical to assume that it would be profitable to approach the subject from the biological side of the problem, which has been long neglected. The physician dispenses treatment in the form of pills and various therapeutic agencies, but behind all this treatment he has the knowledge that tells him how, when, and why to use remedial agents at his disposal. The druggist dispenses the various treatments the physician prescribes. No doubt, in many instances, one could get at a drugstore the same treatment that he receives from a physician, but we consult the physician because of his superior knowledge of living tissue. Does this superior knowledge imply that he can make better pills than the druggist? The physician's superior knowledge pertains to living tissue in its normal and abnormal aspects. It is knowledge of living tissue that justifies the physician; without it he might be simply a druggist. In a similar manner, the prosthetist has at his disposal methods of treatment in the form of bridges and dentures. Are we satisfied to dispense obsolete bridges and plates because they were good mechanically? When we see that the ultimate success or failure of our mechanical treatment rests solely with the vital structures of the mouth, we realize that it is here that we must look for the principles to guide us in determining what is required of our mechanical appliances. The laws of nature are immutable. We must conform our appliances to these laws, or failure is inevitable. The removal of vital pulp to facilitate mechanical retention by means of dowels in the canals is now frowned upon, not because it is wrong mechanically, but because it transgresses the laws of nature. The carving of cusp and restoration of contour were developed through an appreciation of physiological requirements of the mouth. In fact, every great advance in any department of dentistry has been based upon knowledge of living tissue. Such knowledge enables us to determine causes, whereby we may test and weigh the various systems and methods advocated. When we know causes, the treatment becomes simpler and diagnosis more accurate.

This view places the mechanical concept in its proper position: "the treatment, the means to the end." I do not, however, wish to convey the idea that technical skill is not of the utmost importance. On the contrary, I believe that the exacting mechanical heritage we are heir to will always be of inestimable value. It would be just as fallacious to disregard the study and development of technical skill and mechanical principles involved as it is to neglect the biological nature of the problem. It is a question of considering each in its proper perspective, appreciating that both are important, but that the mechanical phase as the treatment is subordinate to the requirements of the living tissues to be treated. Furthermore, the more knowledge we have of the living processes in the mouth, the more we appreciate the need for good mechanical appliances. Therefore, this view will enhance rather than detract from our mechanical technic.

The study of the biological concept in partial prosthesis offers a broader outlook for the future. It is the view we apply to our prosthetic diagnosis. The public little knows the amount of preparation required to develop the knowledge, experience, and judgment necessary to form a conception of the best restoration for any given condition. Even the average dentist does not realize how complicated are the factors that influence the pathological conditions created by the loss of function of even a single tooth. Immediately upon its loss is begun such insidious changes in the occlusion as cause the development of inflammatory conditions that progress until alarming tissue changes may take place, which are akin to those of periodontoclasia, etc. Orthodontists recognize that the correct arrangement of a perfect set of teeth has a vast influence on the proper development of the entire head, and strive in patients at a tender age to approximate this ideal by correcting conditions leading to malocclusion. Periodontists have given much study to means and methods of correcting and changing the occlusal forms of the teeth when age and other conditions contraindicate orthodontic interference.

The time finally arrives when some of the teeth are lost by disease or accident. Then the partial prosthesis expert is called in. With his aid a restoration is designed to prevent further malposition and disturbance, which is sure to follow the loss of any tooth. He must possess the fundamental knowledge that directs the orthodontist and

the periodontist to their conclusions. In addition he must know the laws of mechanics governing the leverage, fulcrum, inclined planes, and the innumerable multiplication of forces in the mouth. Above all, he should have observed over a period of time a manifestation of these influences in the functioning of various conditions. He should then be able to make an intelligent diagnosis and an accurate prognosis.

Natural teeth function in esthetics, mastication, and enunciation. When natural teeth are lost, these functions are impaired. It is our duty to restore them with artificial substitutes. Today, the demands of scientific health service lead the most careful operators to avoid the possibility of infection from devitatilized teeth by extracting diseased teeth, instead of filling root canals. This has resulted in many vacant spaces, and often the teeth most desired for abutments are missing.

It has long been recognized that the fixed bridge, in well selected cases, gives the wearer perfect satisfaction over a period of years. In many other cases, however, failure results in even a few months, for a logical reason that has usually been overlooked. In our effort to prevent failure, we have developed substitutes which, in some cases are very ingenious and worthy of employment, but in others have been injurious to the abutment teeth, very transient in their usefulness, and uncomfortable for the patient. I refer to most forms of clasp removable bridges. There are very many removable attachments on the market, designed to allow a bridge to be removed by the patient for the purpose of cleansing, which is commendable. In many instances the procedure is unsatisfactory, however, for these attachments depend upon friction for the maintenance of position, and if the bridge can be removed easily by the patient's fingers, it often loosens when sticky foods are chewed, causing discomfort. The result is that the frictional member is either so loose as to become bothersome, or so tight as to be virtually a solid fixed bridge. Very few attachments permit movement while *functioning*. One type, called "movable removable bridgework," utilizes a saddle. Its sponsors claim that the saddle in its slight movement in mastication stimulates circulation, and prevents absorption, and that part of the load is carried on the alveolus. Most of the attachments have a bottom or seat that limits the up-and-down move-

ment, and most of them are absolutely as rigid when seated as a soldered tight bridge. It is recognized that each individual natural tooth has a limited movement when functioning, and to prevent that movement results in inflammation of the adjacent tissue, loss of function, over stimulation, breaking down and absorption of supporting tissue.

Some teeth are very firmly imbedded in the alveolus, which has a rich, healthy blood supply. This condition aids in resisting the application of abnormal force. These teeth even adapt themselves to such a restriction in functional movement as is caused by joining two solidly together, as is often done for a short fixed bridge. Some teeth may do this for a long time without apparent harm, but most of them soon show signs of degeneration of supporting tissue. This being a recognized result with the most favorable cases, what is the effect on the supporting tissues of the majority of our cases where the roots are not strong or favorably nourished? Often these weaker teeth must be used as abutments. Therefore, there should be employed some form of attachment that will not place the entire burden upon them, yet will permit them to aid in carrying supplies without irritation, inflammation, or any shortening of their usefulness. For a long time we have watched removable bridgework, hoping for a solution that would prove satisfactory. Many patients have paid large sums of money for, and have gone to great inconvenience in trying, various types of removable work, but they are unanimous in one complaint; namely, "a nuisance to care for." A realization that the present types of partial restoration do not conform with physiological laws, and a search for some means at least of minimizing their harmful results, led to the development of a particular type of attachment with which to anchor restorations to the remaining teeth, and to give the maximum of service with the minimum of objectionable results. This is preferably applied to a type of denture that is anchored to two or more natural teeth and remains in the mouth as a permanent bridge. A return to fixed bridgework seems inevitable. Some of its faults, like the irritation by the facings that used to be common, can now be entirely eliminated with Tinker glazed root-tips and interchangeable teeth. If, in addition, we could incorporate a principle to prevent abutment teeth from being overstrained, we should be a step nearer comfort and

utility. Before we sanction any permanent restoration in the mouth, we must eliminate as many objectionable features as possible.

It is hardly conceivable that, with our present knowledge of infection, rough-ground facings should ever be placed in permanent contact with soft tissue. The new Pontopin facings, made by the Ideal Tooth Co., and Steele's True Pontics solve this problem for those who do not use Tinker pontics. When a saddle is indicated to restore deformity or distribute the load, a glazed porcelain saddle is of course the first choice. A saddle, to function as auxiliary support, cannot be put under permanent pressure without causing absorption. As soon as absorption does occur, the teeth become the sole supports. So a rigid clasp or a tube and post, friction tight, will not allow a saddle to function. No fixed or removable bridge that prevents the natural movement and individual function of an abutment should be indorsed. The articulation should be so balanced and adjusted as to prevent shock to the abutments, or to the opposing or occluding teeth. All this seems logical, if we are to prevent damage to the remaining teeth. What can be done to improve these conditions? It would be folly to enter this discussion unless we could present a remedy. This we hope to do with a new attachment, for use on a fixed bridge, differing materially in principle from many now available for removable work, which are not adapted to remain as fixtures in the mouth, but are intended to be removed daily by the patients.

II. OSCILLATING-ATTACHMENT FIXED BRIDGE

The principle behind the device described below was conceived from a mechanical viewpoint, but also from knowledge of the requirements of living tissue; namely, that *abutment teeth should not be restrained in their physiological movement.* The new attachment referred to has many advantages, among which are these:

(1) It is small, strong, simple, mechanically correct, and not difficult to apply, requiring no expensive or complicated apparatus for its use, nor such accuracy of parallelism as to be beyond the skill of the average dentist.

(2) It can be attached to any style of abutment now used in bridgework.

(3) The abutments can be separately cemented on or in, and finished perfectly.

(4) The bridge itself can be of any type desired, with or without saddle.

(5) It can be temporarily worn without fixation, allowing adjustment over a period of days.

(6) When settling has progressed to the desired amount, fixation at this point controls it.

(7) A limited vertical movement is possible, and the amount is under control.

(8) Variable mesio-distal and bucco-lingual movements are also under control.

(9) It is to all intents and purposes a permanent fixed bridge.

(10) Should breakage occur or inflammation develop, making its removal necessary, it can easily be removed and replaced without mutilation of abutments or pontics, or discomfort to the patient.

(11) When a saddle is used as an auxiliary support, the compression of tissue is under absolute control. When adjusted, the compression is released to the extent that the operator's judgment dictates. Thus it is a "tissue and tooth borne denture" in every sense of the term, allowing the amount of physiological stimulus necessary to maintain circulation and to prevent absorption, a condition claimed by many to be highly desirable.

(12) Last, but by no means least, when this attachment is made of platinum and used in conjunction with an all-porcelain bridge, restorations hitherto deemed impracticable become at once full of promising possibilities, and we rejoice at the opportunity that this type of restoration offers.

These features have never before been attempted in a fixed bridge. They have sufficient merit to demand the attention of all who wish to progress and to promote the best interest of their patients. In a mouth requiring a restoration, the life and health of the remaining teeth depend upon unimpaired nutrition, functional exercise, and cleanliness. These conditions cannot be favored by a restoration that does not allow each individual tooth to have its natural movement and normal stimulation. My oscillating-attachment type of bridge allows functional exercise of abutment teeth, and functional stimulation of

the underlying tissues; and it maintains conditions that are as sanitary as those for the rest of the teeth in the same mouth. When properly constructed it is comfortable, durable, and esthetic, and fulfills all physiological demands (*fig. 1*).

Fig. 2 illustrates the oscillating attachment, which is composed of three parts: an outer part or tube, with a triangular slot running

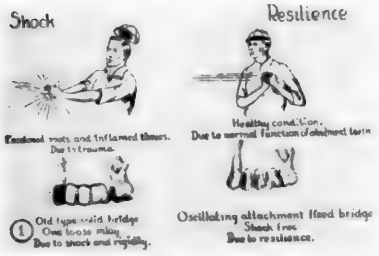


FIG. 1

FIG. 2

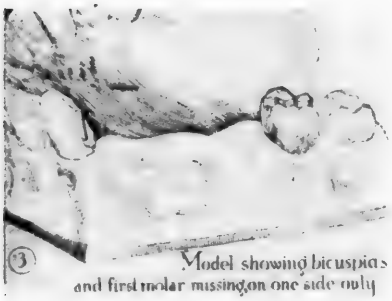


FIG. 3

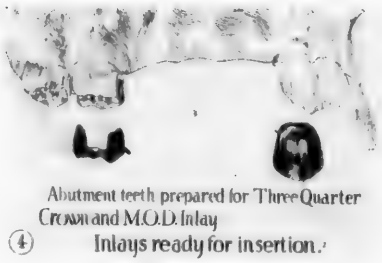


FIG. 4

through it. This portion of the attachment is intended to be imbedded in the bridge proper; and, when completed, becomes continuous with the pontics or supplied portion of the restoration. Through this triangular slot is loosely fitted a triangular rod with a flange easily soldered to the inlay, crown, or other anchorage to the abutment teeth. When these two parts are properly assembled, they are held together by a unique screw or lock pin. These oscillating attachments

are made of hard platinized gold for regular use, and of iridio-platinum for jacket crown and other porcelain construction.

The method of preparation of abutment teeth and types of anchorage must be governed by the conditions of each individual case. This type of work has been found as successful as any type of inlay or crown heretofore used for fixed bridgework. In fact, there is less strain on the abutment teeth. Therefore, inlays are not as likely to

FIG. 5

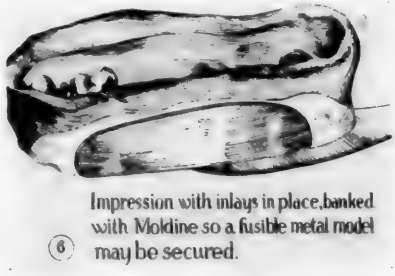


FIG. 6

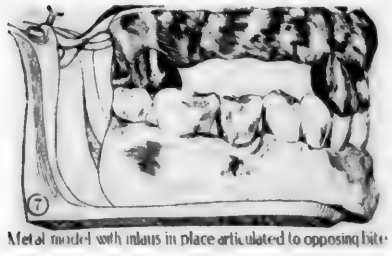


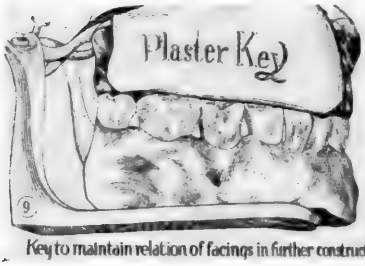
FIG. 7

FIG. 8

be dislodged. Deep cutting is avoided, as the attachment is usually set outside of the cavity, although it may be reversed if desired. Presupposing the teeth to be vital, and in normal position and alignment, the abutments are prepared for the Carmichael and M.O.D. inlay (*fig. 3*). The completed abutment inlays should be placed in their respective positions in the mouth (*fig. 4*), and a bite for articulation secured. Then take a plaster impression, which should remove the abutment inlays. If they do not come away with the impression,

they must be carefully replaced therein with great exactness (*fig. 5*). After assembling the impression, surround it with Moldine (*fig. 6*). When crowns are used for abutments, their inner undercuts should be eliminated with the same material. Pour into this impression a low fusing metal, such as Ney's Dialloy or Bechtech Plastic Metal. No separating medium is necessary. When the metal first becomes crystalline, pack it down with light pressure, to insure a model with clear cut details. The metal model, now cooled and with impression removed, produces a model from which the abutments may be readily removed and replaced with accuracy (*fig. 7*). Mount the metal model with an occluding cast and articulate anatomically (*fig. 7*).

Select the type of teeth to be used for pontics, and grind them to



Key to maintain relation of facings in further construction

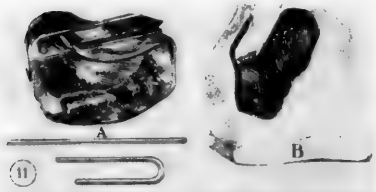
FIG. 9

Reflection showing advantage of the plaster key in allowing pontics to be removed for backing, etc.

FIG. 10

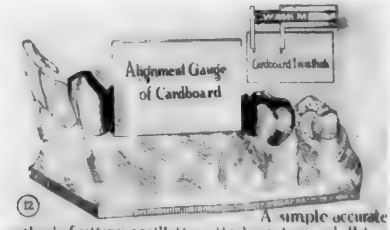
fit the alveolar ridge and for general shape, size, and alignment (*fig. 8*). All surfaces thus ground should be super-glazed before setting the bridge. Oil the labial or buccal surfaces of the model, and build a plaster support or key (*fig. 9*) against the faces of the abutments, waxed-in pontics, and adjoining teeth on the model, thus retaining the assembled relation (*figs. 9–10*). Adjust the oscillating attachments in position on the abutment inlays. Grind the shape of pontics on the lingual portion so that the attachments fit in the desired position, which should be about at contact point, and stand vertically (*fig. 10*). Grind or file the abutment inlays so that the oscillating attachments touch on its entire length. The oscillating attachments themselves may be altered considerably without weakening them. Do not grind them too short. Leave them as long as the bite will allow. Grind the

attachments at both ends so that the screw hole remains in the center. Also grind or file at the cervical end to produce the natural interproximal space. When the attachments seem straight and parallel to the eye, wax one to its abutment inlay. It is important that the attachment be most carefully waxed in exact position, and no space left where anti-flux or investment can enter between the base of the triangular part of the attachment and the abutment inlay. Remove the screw of the attachment and slip off the outer tube. Carefully



Triangular part of oscillating-attachment waxed to inlay, with wire support. Same case invested, ready to solder.

FIG. 11

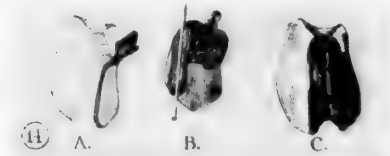


A simple accurate method of setting oscillating attachments parallel to each other, and in same plane or alignment.

FIG. 12

Completed oscillating attachment soldered to Inlays

FIG. 13



Outer part of oscillating attachment waxed to modified backing of pontic, with wire support while soldering

FIG. 14

coat the entire exposed surface of the portion of the attachment that is waxed onto the inlay with anti-flux. Bend a small wire in the form of a staple, and slip it over the edge of the inlay to hold the attachment in place while soldering (*fig. 11, A*). Then only a small amount of investment is necessary. Make certain that the soldering space is open enough, and keep the entire investment hot when soldering to prevent burning. Avoid the use of too much flux, or the edges of the investment and the antiflux will be destroyed (*fig. 11, B*).

After soldering, clean and replace on the model. With any type of parallelometer, set the second attachment exactly parallel to the first (*fig. 12*). Wax in place, invest, and solder as in the case of the first

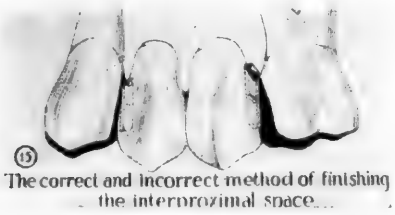


FIG. 15

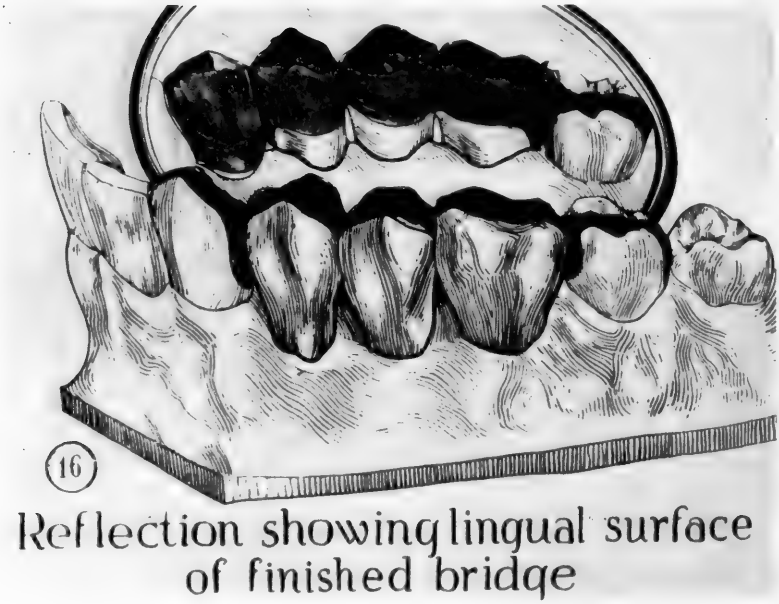


FIG. 16

attachment and inlay. Place on the model the abutment inlays thus completed, and replace the outer tube portions of the oscillating attachments on the triangular portion, and set the screws in place (*fig. 13*).

Prepare the backings for the pontics, and file or grind them sufficiently so that the pontics occupy their original positions (*fig. 14, A*). Wax the tube or outer portion of the oscillating attachment to the backing adjacent to it, but do not wax the individual backings together. Remove the screws, lift off and remove facings. Use the same pre-

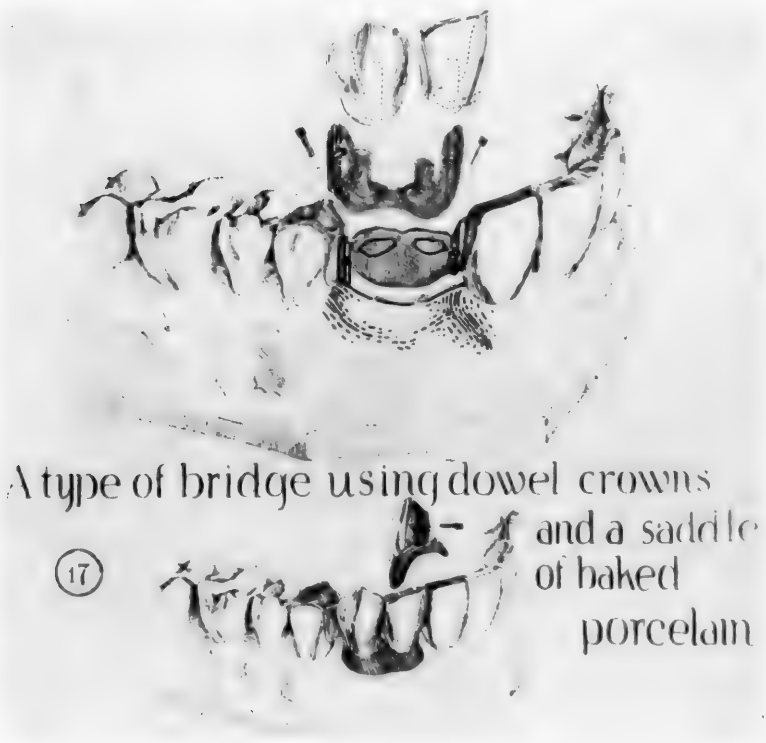


FIG. 17

cautionary measures in investing and soldering these portions. Apply anti-flux very carefully to the inside slot, and be certain to fill the screw hole and threads. It is well to place in the screw hole a short piece of carbon (lead from an "eversharp" pencil). A short piece of wire placed in the triangular slot serves to hold the attachment more firmly in the investment (*fig. 14, B*). With care in investing, boiling

out the wax, thorough technic in fluxing, and complete control in soldering, no difficulties should be experienced.

Invest each backing with its attachment separately, and solder with 20 k solder, restoring the lingual contour and occlusion of each tooth completely. Remove from the investment, clean and re-assemble on the model. The backings, after being built up with solder, should require a little filing or grinding at the contact point to fit exactly, so that the least amount of solder will unite them and prevent shrinkage. The lingual occlusal surface for each pontic may be cast instead

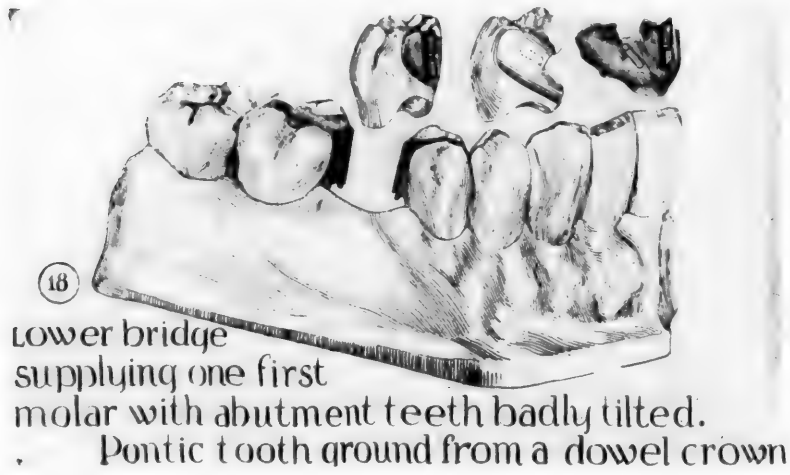


FIG. 18

of soldered if preferred (*fig. 14, C*). Wax these contact points, remove the assembled parts from the model, invest, and join with 20 k solder. Clean thoroughly, cement the facings in place, finish and polish, assemble on the original model, and place screws of the attachment in place. The cervical movement of the supplied teeth may be adjusted by widening slightly the slot in the triangular portion of the attachment. The bucco-lingual and mesio-distal movements may be increased by slightly filing the face of the triangular portion toward either end, never at the center (*fig. 2*). Carefully finish self-cleansing interproximal spaces (*fig. 15*).

By carefully following the technic as outlined, a fixed bridge is produced which fulfills the exacting requirements of an ideal restoration (*fig. 16*). *Figs. 17*, *18*, and *19*, are finished cases of this type of bridge-

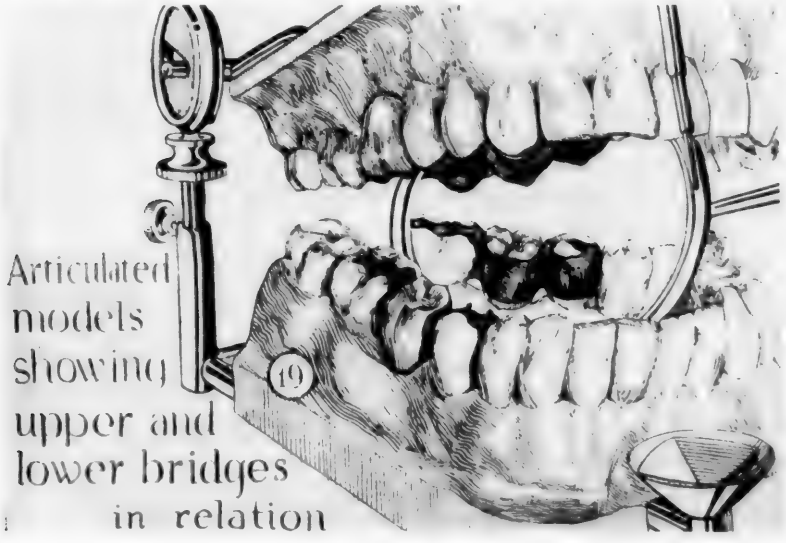


FIG. 19

FIG. 20

work. With special iridio-platinum oscillating attachments, it is possible to use a reinforced porcelain jacket-crown as anchorage, with perfect satisfaction in any all-porcelain restoration (*fig. 20*).

III. PRECAUTIONS IN MAKING THE NON-FRICTION OSCILLATING ATTACHMENT

The features of operation demand accuracy of technic in construction. The following summary will help beginners to avoid failures, these conditions being regarded as essential:

(1) A soldering investment that will stand the heat and not burn up at the thin edges Most of the high-heat inlay investments will answer, if care is taken to avoid fluxing the investment.

(2) A sticky wax that will hold the parts securely and not leave carbon on the surface when melted away. Parr's fluxed wax, made by S. S. White Co., is ideal.

(3) A good anti-flux to paint on all surfaces where solder is not wanted. Steele's anti-flux, rouge ground fine and mixed with alcohol, drawing ink (Higgins' India Ink), are all good.

(4) Good or bad flux is often the answer to good or bad soldering. A lump of unslaked borax moistened with water, rubbed to a cream on a ground-glass surface, is the best and only flux needed.

(5) The working qualities of the solder is of great importance. S. S. White 20 K is strongly recommended.

(6) The blow-pipe may be the cause of failure in soldering. "Know your blowpipe;" be sure the valves are under control, and capable of brush and pointed flame adjustment.

(7) Cleaning the soldered piece to get rid of the flux is important. Ten per cent sulphuric acid, or a saturated solution of alum, may be used. Small work may be boiled in a test tube.

IV. SUMMARY

The main features of the oscillating attachment are these:

(1) An attachment that allows normal function of the teeth to which the restoration is anchored.

(2) Allows enough lateral movement to the pontics to absorb the shock of sudden force.

(3) Allows enough vertical movement of a saddle to cause compression and relaxation of the mucous membrane.

(4) Small, strong, universally applicable.

(5) Easy to attach to inlays and to pontics.

(6) Easy to assemble in the mouth.
(7) Possible to adjust to individual conditions.
(8) Comfortable and sanitary in use.
(9) Easy to dis-assemble for repairs or corrections.
(10) No expensive outfit required for its use.
(11) No extra skill required to make or insert it.
(12) No special care or caution required of patient.
(13) No interference with esthetics or anatomical perfection.

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By

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